CYSTIC DISEASES OF THE KIDNEY

chiefly in relation to

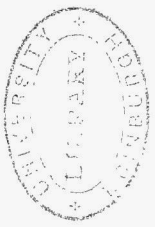
THE LARGE POLYCYSTIC KIDNEY.

GRADUATION THESIS

by

JAMES RITCHIE M.B.: M.R.C.S.Eng.

1891.



Note

The numbers enclosed in brackets relate to Bibliographical references at the end.

The numbers in the margin refer to Plates in the volume of Illustrations, and to Microscopical preparations, in a separate volume.

Annexed to each plate is reference to the microscopical preparation from which the drawing was made.

The numbers on the microscopical preparations which relate to the text are in red ink. The numbers in black were used for laboratory purposes and for reference.

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In addition to that cystic condition of the kidney and its pelvis, and sometimes also of the ureter, which we know as Hydronephrosis, cysts occur in relation to the kidney of at least five different varieties.

I. Scattered cysts in kidneys otherwise apparently healthy, often these cysts are small, occasionally of large size.

2. Scattered cysts in Cirrhotic Bright's disease.

3. Congenital cystic degeneration of the kidneys.

4. General cystic degeneration of the kidney in the adult - the large Polycystic Kidney.

5. Hydated cysts.

In the following paper although the first four of these varieties will be described, it is intended to enter into a careful discussion of the fourth variety only, viz, the large Polycystic Kidney of the adult, and to consider the three previous varieties mainly in relation to the fourth.

In studying the literature of the subject there have been found many cases, both of Hydronephrosis and of single cysts,

which are described as cystic kidney.

Hydronephrosis.

I shall enter at some length into this subject because of a case which came under my observation some time ago, and which presented signs and symptoms which led to an error in diagnosis, and I shall afterwards have occasion to refer to the case both under etiology and under diagnosis.

Mrs L. age 48, of rheumatic constitution, had borne ten children, for some years she had been anemic, had lost flesh and strength and was liable to paroxysmal attacks of pain in the loins, specially on the left side, accompanied by great prostration and sickness, occasionally preceded by a copious flow of urine.

There was no history of gravel nor of acute inflammation of the kidneys, or of the genito-urinary tract. During the twelve or fourteen years I acted as her medical adviser she did not suffer from any of the ordinary causes of hydronephrosis, except several pregnancies.

Three years before her death there was found in the left hypochondrium a nodular, elongated, firm mass, slightly moveable. In July 1889, about nine months before the fatal occurrence, during a paroxysmal attack a considerable amount of blood appeared in the urine, this lasted only for a few hours

after which the urine presented its usual characters, viz, clear, sp. gr. about 1012, acid, and always contained a trace of albumen, but no tube-casts were discovered. At the time of this illness the right kidney was found to be slightly enlarged with a nodular surface, and as the patient was thin the nodular condition of the left kidney was clearly made out, and the organ was decidedly enlarged. For years there had been a mitral systotic murmur, and this still remained but in a less degree than formerly. The parozysmal attacks from which she had suffered at intervals were very severe, causing great prostration, confining the patient to bed for several days on each occasion. They were always accompanied by pain in the back and specially in the left side. From July till the fatal illness she had no severe attack. In December after having been shopping she was taken suddenly ill, became quickly unconscious and died within 2 hours from cerebral hemorrhage.

On post mortem examination the bladder, uterus, with its appendages, and the ureters presented no evidence of disease. In each flank there was seen a nodular swelling, that on the left side being large and most marked. The kidneys were found to be surrounded by much connective tissue and were much more adherent than usual. The left kidney (Pl. I) presented on its surface several rounded elevations resembling cysts having kidney substance between them, the pelvis was

much dilated, and no fluid could be made to pass from the cut end of the ureter even when pressure was made on the dilated pelvis. On section 150 grammes of clear straw colored urinous fluid escaped, this contained about 1 per cent of urea, 2 per cent of albumen, and chlorides in abundance. The calyces were found to be dilated into sacs, and formed with the dilated pelvis one large cavity. The dilated calyces had reached the surface of the kidney and appeared there as rounded elevations with clear membranous walls, and as already noted there were portions of kidney substance between them. The ureter passed obliquely in contact with the dilated pelvis for some distance, it was completely occluded, and even the most careful examination of the inner surface of the pelvis failed to shew the smallest orifice leading into the ureter. The lining membrane of the pelvis seemed to be continuous over the ureter.

The right kidney shewed a similar condition to the left in a slight degree, its pelvis was much dilated, and the orifice of the ureter narrowed. There were no calculi. The heart was much hypertrophied, the wall of the left ventricle very much thickened.

× Sections L.Rt 1 L.Rt 2

On microscopic examination of the right kidney (×) it is found that the capsule is slightly thickened, there is great engorgement of the capillaries, and dilation of the

veins, the walls of the arteries are thickened, the intertubular stroma is not much increased, in certain areas a considerable infiltration with leucocytes is observed, and an increase of the connective tissue corpuscles, some of the glomeruli are also infiltrated. The secreting epithelium is in many places much changed the nuclei straining badly or not at all.

The left kidney is much more profoundly altered (* Sections I. 3 to 7). The capsule is much thickened, the secreting structure in certain areas is entirely replaced by connective tissue, the walls of the vessels hypertrophied, the veins dilated and in some places irregular forming pouches, the capillaries empty or with little blood. In some areas there is a copious infiltration with leucocytes, the tubes and Malpighian bodies have entirely disappeared from some localties, a few tubes are dilated near the cortex in those regions which are least changed, but a point of great interest is that many of the glomeruli have undergone colloid change (Pl. II ** Pl.II Fig 1.), and there may be seen every stage of the process from slight alteration up to entire colloid transformation. Even those bodies in which the glomeruli are least changed have the capsule much thickened.

The etiology of this case seems to be, interstitial nephritis, with paroxysmal congestive attacks, Ureteritis descendens leading to narrowing of ureters and ultimately

obliteration of the orifice on the left side, and Hydronephrosis.

Jaccond lays great stress upon the intermittent character of the cirrhotic process - whether it affect liver or nerve centre, in this case the intermittent character is very prominently brought out by those paroxysmal attacks from which the patient suffered, accompanied by pain in loins, copious flow of urine, and on one occasion by haematuria. In cystic kidney these symptoms are often well marked and in this case the symptoms taken in conjunction with the tumors found on examination, led to an error in diagnosis, the case having been supposed during life to be one of large Polycystic kidney not very far advanced.

Before leaving the subject of Hydronephrosis I wish to draw attention to some sections of kidney from a newly born lamb which was affected with Hydronephrosis of both kidneys (*) and also to some sections from a young pig four months old, one of the kidneys of which was considerably dilated, and the kidney substance in consequence was very much thinned. (**). The sections from the pig are made through the whole thickness of the wall of the kidney the greater part of which was of about the same thickness as the portions shew. These exhibit to how great a degree the kidney structure may be subjected to pressure without the

* Sections La. 10411

** Sections Pig 849

tubules being obliterated, doubtless because the capsule yields somewhat readily to slowly increasing pressure. The parts most seriously altered are those immediately under the capsule and those next to the pelvis and calyces, in which region there is a great increase of connective tissue. The othere points are that the character of the epithelium is not the same throughout being in one part cubical, in another columnal or spindle shaped, and that the result of the change has been to cause a great proliferation of epithelium at one portion. The latter change is well seen in one section (*).

*Section Pig 8

I. Scattered Cysts in Kidneys otherwise apparently healthy.

These cysts are often small, occasionally they are so large as to contain a pint or more of fluid. The wall of the cyst may be thin or thick, and is lined with squamous or cubical epithelium. The contained fluid is usually clear, having in it the elements of urine. The surrounding kidney structure is altered in variable degree in proportion to the rate of growth of the cyst and the amount of pressure on the adjacent structure.

The pig is very liable to this kind of cyst. In the specimens which I have examined (**) the cysts have been lined with squamous or cubical epithelium, in 12 there is a small cyst in which there is a great proliferation of the

** Sections Pig 1 № 12 Pig 2 № 13 9 14

epithelium, which is seen lying in several layers. The wall of the larger cyst contains much connective tissue, and the adjacent structures are displaced by pressure. The cysts were seen bulging on the surface of the kidney.

2. Scattered Cysts in chronic Cirrhotic Bright's disease.

These are usually small in size but are occasionally found as large as a grape. They are sometimes numerousiin the cortex, the Malpigian bodies often have the capsule dilated, and occasionally in the cones a moniliform sacculated dilatation of the uriniferous tubules is to be seen. Their contents are often clear, but occasionally blood and colored matter are found.

Grainger Stewart (34) states that some originate in the capsules of the Malpighian bodies, and the compressed group of vessels may be seen on one side, that some result from obstruction of tubes and consequent dilatation of the distal portion, that some again are produced from morbid growths of epithelium elements. Hertz (91) Klein (27) Beckmann (16) and many others have studied these cysts. It seems to be the general opinion that they are produced from the Malpighian bodies or from them and a part of a tube. Wilks and Moxon believe that the organs are badly formed.

Cysts are found not only in the later but also in the early stages of Cirrhoris of the Kidney. Microscopic examination reveals the changes found in cirrhosis, an increase of the connective tissue, (*) many of the Malpighian capsules greatly thickened, some of them slightly dilated with the glomerulus compressed in varying degrees (**) and in many the glomerulus may be seen in various stages of colloid degeneration, a few of the tubes dilated, many atrophied some filled with colloid matter. In the specimen "Br 81" 35 there are seen a large cyst and a small one, from both of them the epithelium has been shed but here and there portions of a flattened cubical lining are seen. Immediately around the cyst the ordinary renal elements are more altered than in other regions, and round about a third part of the cyst wall there is a great amount of connective tissue, and where this passes into less profoundly altered structure it can be noted that this fibrous band has, at one time, contained many tubules the contents of which have been absorbed, leaving here and there only a few epithelial cells and small spaces as vestiges of former structures.

* Sections 28 to 36

** Sections 34. 36. etc

In the majority of these cysts the epithelium is flattened, No 29 & 31 in some however it is cubical (***), and occasionally this is found in more than one layer and undergoing colloid change (**).

*** Sections 33 small cyst at side of it. 33

33. 34. 36

These conditions are commonly seen in Interstitial Nephri-

tis with cysts. In all the specimens marked "Ca", which are portions of a somewhat enlarged kidney, with many cysts, there is a considerable development of hyaline connective tissue, running in somewhat definite bands, very well marked under the capsule, and radiating in several directions, containing many nuclei which strain well. In all the regions in which this tissue is found the changes in the secreting structure are more marked than elsewhere.

The conditions, then, under which we find the kind of cyst which we are now considering are as follows. They occur in organs giving evidence of considerable local irritation, and in individuals in whom the metabolic process*es* are badly performed, the morbid products being eliminated by the kidneys. These cysts are few or many, generally small, the presence, in some of the smaller of them, of the remains of a glomerulus along with the granular contents which have compressed it give a sure indication of the nature of the part observed, they are found mainly in the cortical substance - that is to say, where the Malpighian bodies most abound. In the normal kidney, corpuscles are often seen in section in groups of two, three, or four, and occasionally two are found in close juxtaposition. It will be found on examination of the sections* of foetal kidneys that they are developed in pairs. In some sections it is evident from the position of a small cyst (**) that,

* Sections 21.23.24.27 etc

** 72.73.34.36

there is in many cases a marked increase of connective tissue at this part, other capsules in the same sections are thickened very considerably and embrace closely a shrunken glomerulus, giving the impression that this new tissue has encroached upon it. Furthermore in other organs of the body we find that irritative changes are more common at the orifices of the organ than elsewhere, now the neck of the Malpighian capsule is described as contracted, and it is at this part that the epithelium changes from squamous to cubical. Possibly then, for these two reasons, in consequence of changes around the neck of the tube and within it, stenosis or occlusion is more likely to occur at this locality than elsewhere.

3. Congenital Cystic degeneration of the Kidneys.

The diseased organs present some points of resemblance to those found in cystic degeneration in the adult. The kidneys are enormously increased in size so as to form a serious embarrassment to delivery as in the cases reported by Cormack (4) and Siebold (14). On section the kidney is found to be transformed into a multitude of cysts of various size, often the size of peas in a matrix from which they can be readily detached, and either no secreting substance, or very minute portions of it, can be discovered. Dr Gull in a case which he examined could not find any secreting substance.

This affection has been studied by many. Othmar Reer (92), Littré (94), Virchow (24 p267), Förster (93), Hertz (91) Duffey (29), Klein (27), Rosenstein (75) and others.

Rindfleisch (36), holds that congenitalcysts originate in precisely the same way as the cysts resulting from interstitial nephritis, that they invariably start from the Malpighian bodies, that the uriniferous tubules may also undergo cystoid degeneration in their continuity but such cysts are always formed by the dilatation of a single tube.

Virchow holds (24) that it owes its origin to a foetal inflammation of the renal papillæ, causing atresia of the papillae, so that there is no communication between the uriniferous tubules and the calyces of the kidney.

Köster (32) believes that it is due to abnormal development, absence of renal calyces and pelvis, and in many cases of the ureters also. In favor of this view there are two circumstances - First, that, as Kupfer (95) has pointed out, the blastema for the tubuli uriniferi is developed by itself, independently of the evolution of the Wolffian duct. Second, other malformations are very common in the subjects of Congenital cystic kidney.

With the view of testing Kupfer's statement I have examined by serial sections the urogenital structures of a foetal

× Pl. III Fig 1.2. rabbit of the twelfth day of development.× The kidneys of human foetus 1) One inch and three eighths long, 2) one inch and three quarters long, 3) Three inches long. 4) Three inches and five eighths long, 5) Four inches and one eighth long, 6) Five inches and three quarters long.

In the earliest stage I have not been able to trace any pelvis (××). In the next specimens (××) there is a well marked pelvis which seems by a process of budding to grow into the kidney substance forming what are afterwards the calyces. But there is no marked developed of uriniferous tubules until between the third or fourth month (×××), and even then they are very rudimentary. In the oldest foetus examined, probably between the fourth and fifth months, the collecting tubules can be clearly seen opening into the calyces.# If there were a failure in developement, however induced, of the communication between the secreting structure and the pelvis or calyces the result would be cystic kidney.

×× Sections F.R. 15

×× Pl. II Fig 2 Sections 17 & 21

××× 22. 23

26. 27

The malformations commonly found in conjunction with this disease, are absence of the pelvis of the kidney, or malformation of it, of the ureter, bladder, and urethra, also hare-lip, and club-foot.

The contents of the cysts - urates, hippuric acid, etc- shew that they have been produced by urinary secreting structures.

Cystic disease of the liver is a common concomitant of the large cystic kidney in the adult, but, as Lancereaux first

pointed out, it has never beenfound associated with congenital cystic kidney.

IV. General Cystic Degeneration of the Adult Kidney. The Large Polycystic Kidney.

The following is the clinical history of a case which I had under observation for several years.

Mrs Fo age 45 at the time of her death in 1888. Between eight and nine years previously, after a confinement, while endeavoring to grasp the uterus to control hemorrhage, I noticed to the left of that organ a firm lobulated mass, which was moveable, and as far as could be made out was not connected with it. There were no symptoms, I did not think of the kidney, and it was only after the condition was further advanced, that a diagnosis was made. At a subsequent confinement I found that the tumor had enlarged, and on more careful examination it was found that a similar swelling existed in the other flank. On bimanual palpation the tumors moved with the kidney and were evidently altered kidneys. The urine was copious pale, low sp. gr, but it contained no albumen. For about two years before her death Mrs F. lost flesh, became pale and greyish in complexion. About a year before the fatal termination she consulted me in consequence of sickness and constipation, she was then very thin so that not only could the character of the renal tumors be made out, but some of the individual cysts could be distinguished. The lower end of each tumor passed down into the iliac fossa. The urine was very copious, a medium sized

chamber pot being filled over night. After this attack she was again able to attend to her household duties, but was in very poor health. When again she had to seek advice at the end of October 1888, rather more than a month before her death, it was found that she was suffering in a similar way to that which she had done about a year before, but this time more severely, and thirst was now a very prominent symptom. The tongue was coated, the bowels constipated, chiefly because so little food was retained. The urine was very copious, although not so much increased as it had been a year previously sp. gr. 1006 a trace of albumen, no tube casts. Notwithstanding the greatest care as to rest, warmth, and easily assimilated nourishment there was no real improvement, the sickness was less severe, but the appetite remained very poor, strength was lost, the breathing became laboured, not because of oedema of the lungs, there was no oedema, the air entered freely at every part, but evidently the changes between the blood and the air did not take place. Emaciation was very great. For a day or two before the end the breath was quite cold, there was no convulsion, consciousness was retained to the end, the patient died of exhaustion.

Post mortem examination was made about twenty hours after death. Examination of the abdomen only could be got. The body was much emaciated, cadaveric rigidity was well marked. On opening the abdomen it was evident that there was a large

very irregular mass in each flank. There was no gross lesion of any of the abdominal viscera. On endeavouring to remove the kidneys in the usual way, it was found that they were much adherent than usual in consequence of a greatly increased development of connective tissue.

On examination after removal it was found that the right kidney measured 8 inches long, 3 1/2 broad, the left was a little larger. After injection with gelatine and carmine the left one weighed 41 oz, the right one 27 oz. After removal of a considerable amount of connective tissue each organ formed an elongated irregular nodular mass, which seemed to be entirely transformed into cysts of all sizes and colors.* The hilum was nearly hidden by the cysts. The smaller kidney was bisected in the long axis, after section the calyces could not be recognised, the greater part of the organ - medulla as well as cortex seemed to consist of a mass of cysts of all sizes, imbedded in a firm fibrous stroma, here and there, specially near the cortex, were islands of tissue evidently kidney substance.# It is to be noted that the medulla was as much altered as the cortex, indeed it was near the cortex that the greatest amount of kidney structure was seen.

*Pl. IV.

#Pl. V.

The contents of the cysts were very varied as to color and consistence, in some fluid in others semisolid, some light straw colored, others reddish and various shades of brown. The clear fluid contained a notable amount of albumen. A reddish fluid

from one of the cysts contained 1 o/o of urea, another cyst had rather more than 1 o/o.

In the following pages will be found a précis of 88 cases of this affection which I have been able to collate. In addition to these there are several the records of which I have not been able to peruse.

	Date	Sex	Age	Name	Reporter	Previous Illness	Symptoms.
1	1822	M.	49		Boyer	Paroxysms of lumbar pain.	Lumbar pain and vomiting.
2	1840	F.			Tairgnot Michalowicz		Convulsions.
3	1841	F.	39	M.A.B.	Rayer	Uterine Cancer, 2 years.	Cachectic.
4	1841	F.	aged		Rayer		
5	1841	M.	49	G.R.	Rayer	Lumbar pain, 10 years.	Bloody stools.
6	1841	M.	48	Finot	Rayer		Dyspnoea.
7	1841	F.	29	Mme H.	Rayer	Convulsions a week ago.	Convulsions twice after.
8	1847	M.		Geo.H.	Coote		Paralysis.
9	1850	M.	46		Hare	Blood in urine 9 months ago.	Convulsions.
10	1854	F.	68		Josse	Haematuria a year ago.	Pneumonia at right apex.
11	1854	M.	41	H.	Conway Evans		

Albumen	Blood & oedema	Sp.gr. etc.	Tumor	Cause of Death	Organs Cystic	State of Heart.
			Left hypochondrium	Incomplete Apoplexy.	Kidneys	
				Coma	Kidneys and Liver	
		Scanty Dark		Coma	Kidneys	
				Coma	Kidneys	
	No oedema	Normal quantity		Delirium	Kidneys	
None		Normal		Convulsions, Inflammation of Lung	Kidneys	
				Convulsions and Coma	Kidneys	
				Cerebral Haemorrhage	Kidneys	Hypertrophied.
1/10		1008		Uraemia	Kidneys	
Considerable	Blood once.	On one side.			Kidneys	
None				Heart	Kidneys only 1 very large.	Hypertrophy & Dilatation

	Date	Sex	Age	Name	Reporter	Previous Illness.	Symptoms.
12	1855	M.	Aged		Bourier		
13	1855	M.	40		Gray	Blow on back Haematuria 7 years ago.	Thin, anaemic. vomiting.
14	1856	M.	53		Bristowe	Pain in epigastrium and right side 10 weeks ago	Haematuria.
15	1857				Blachez		Pain in Abdomen.
16	1858	F.	54		Bristowe		
17	1858	M.	48		Markham	Supposed good health.	Sudden Coma.
18	1858	M.	43		Lancereaux		Drowsiness, vomiting, Dyspnoea.
19	1860	M.	50	H.L.F.	Hogg	Obscure urinary symptoms 7 years ago.	Pain in renal region, vomiting, gouty.
20	1860	M.	30		Bright	Haematuria and Albuminuria 2½ years.	Emaciated, feeble, frequent haematuria.

Albumen	Blood & oedema.	Sp.gr. etc.	Tumor	Cause of Death	Organs Cystic	State of Heart.
				Dilatation of Aorta.	Kidneys	Hypertrophied.
Some	Blood often.			Pulmonary Oedema.	Kidneys	Normal.
	Blood			Asthenia	Liver & Kidneys	Much Hypertroph.
None					Kidneys	
					One only	
Abundant				Cerebral apoplexy, Coma	Kidneys	Hypertrophied Vessels atheromatous
Slight				Coma	Liver, Thyroid Kidneys	Hypertrophied.
				Exhaustion	Kidneys 1 large 1 small	Hypertrop. Fatty, large.
Some	Some		First on one side then on other	Convulsions & Coma.		

	Date	Sex	Age	Name	Reporter	Previous Illness.	Symptoms.
21	1861	F.	65		Frerichs		Pneumonia
22	1863	M.	50		Lancereaux		Wound of limb.
23	1867	F.			Jaccond	No history	Taken to hospital unconscious.
24	1867	F.	39		Chantreuil		Phthisis.
25	1868	F.	40	S.J.	Church	Hemiplegia 3 yrs. ago, 5 days ago a fit.	Recent fit, sub-comatose.
26	1868	F.	46		Joffroy		Cachectic, thin.
27	1868	M.	50	G.	Lancereaux	Dyspnoea 8 months diarrhoea 3 "	Intense dyspnoea.
28	1868				Bousseau		
29	1869	F.	74	Marie R.	Leboucher	Oedema of lower limbs for 2 yrs.	
30	1870	M.	48	Wm.A.	Whipham		Bronchitis.
31	1870	M.	49	Ed.S.	Whipham		Dyspnoea, bronchitis.
32	1873	F.	43		Grainger Stewart		Symptoms of chronic Bright.
33	1874	M.	52		Lorey		Gouty, epistaxis.
34	1876	M.			Harris		Sudden unconsciousness paralysis on right side.
35	1876	M.	29		Laveran	Loss of flesh for months.	Cachectic, thin, earthy complexion

Albumen	Blood & oedema.	Sp.gr. etc.	Tumor	Cause of Death	Organs Cystic	State of Heart.
				Pneumonia	Liver & left Kidney	
	No oedema.			Uraemic Coma	Kidneys	Hypertrophied.
				Coma	Kidneys	
				Pneumonia of both bases.	Liver and Kidneys	
				Cerebral haemorrhage	Kidneys	
					Liver and Kidneys	
	Never oedema			Cerebral trouble	Kidneys	
	Oedema		In epi-gast-rium.		Liver and Kidneys	
Con-sider-able. 1/5	Blood no oedema	pale		Pulmonary oedema	Kidneys	
	No oedema			Asthenia and asphyxia	Kidneys	Hypertrophied.
Trace		Copi-ous low sp.gr.	In both flanks.	Uraemia	Kidneys	
Notable		Copi-ous.		Lucid to the end.	Kidneys	
Trace		1016 pale		Coma	Kidneys	Hypertrophied.
No Kidney trou-ble.			None	Sudden death in bed.	Kidneys	Healthy.

	Date	Sex	Age	Name	Reporter	Previous Illness	Symptoms.
36	1876	M.	23		Laveran	Ill 2 months	Chest trouble, nothing pointing to disease of kidneys.
37	1876	M.	48	G.F.	Harris	None	Sudden unconsciousness
38	1876	F.	45	Ad.B.	Michalowicz	Fell down stairs 3 years ago pain in loins, haematuria a month later and recurrence.	Loss of flesh vomiting.
39	1877	F.			Rose		
40	1877	F.	5½	H.H.	Talamon	Tumour from age of 6 months.	Croup, Tracheotomy.
41	1877	F.	45	L.M.	Lataste		Thin, feeble, vomiting, dyspnoea.
42	1879	M.	52	G.F.B.	Deaver		Dyspepsia, dyspnoea.
43	1879	M.	37		Aman & Axel Key	Renal calculi 1871-72 pain in loins since.	Cold.
44	1880	M.	40		Hare	Haemorrhage & tumour 9 months before.	Sudden unconsciousness
45	1880	M.	40	S.M.	Gairdner	Repeated haematuria since age 18.	
46	1880	M.	38	J.C.	Chappnis Bond	Frequent micturition for several years.	Headache, singing in ears constipation, loss of flesh.

Albumen	Blood & oedema.	Sp.gr. etc.	Tumor	Cause of Death	Organs Cystic	State of Heart.
no kidney trouble.				Pulmonary phthisis	Kidneys	
trace		1016 pale		Cerebral haemorrhage.		Hypertrophy & dilatn. arteries atheromat.
none		dark with deposit	Right flank	Cerebral haemorrhage	Liver & kidneys.	Slight Hypertrophy.
					Ovary & kidneys .	
no kidney symptoms.			Since age of 6 mos.	Croup.		
	bloody	400 gms.		Coma	Liver & kidneys	Notable hypertrophy.
none	oedema			Pneumonia & pleural effusion.	Kidneys	Dilated.
much		scanty		Uraemia	Kidneys	
1/10	sometimes	1008	On left side	Uraemia	Left advanced right commencing.	
	5 times		Both sides, left easy to find.	Uraemia	Kidneys	Dilated
some	twice	1014 30 oz	Left side	Coma	Kidneys	

	Date	Sex	Age	Name	Reporter	Previous Illness	Symptoms
47	1880	M.	47	Wm.S.	F.Eve	Haematuria for a week, Epistaxis	Considerable dyspnoea
48	1880	F.	37	H.B.	Duboc	Pain in loins & nausea for a month	Headache, sickness, loss of flesh
49	1880	M.	50		Brigidi & Severi	Almost constant pain in loins for some years.	
50	1881	F.	52		Duguet	Headache	Headache & sickness.
51	1881	M.	65	P.V.	Chotinsky		slight oedema
52	1881	F.	51	C.R.	Strubing	Pain in loins 10 years before, blood in urine.	Pain, loss of appetite, constipation, sickness.
53	1881	M.	40	H.S.	Clarke	Ill from time to time for some years.	Loss of strength, cachectic.
54	1881	F.	67	A B.	Juhel-Renoy	Sudden oedema 2 months ago. Haematuria.	Dyspnoea, speech gasping.

Albumen	Blood & oedema	Sp.gr. etc.	Tumor	Cause of Death	Organs Cystic	State of Heart
2/3	blood no oedema	1010 pale		Coma	Kidneys	Hypertrophied
present	no oedema			Coma	Kidneys Liver & kidneys	Hypertrophied
none		low pale	Both sides	Coma	Kidneys	Hypertrophied
little	oedema	1000 200 grms.	Right side	Convulsions & Coma.	Kidneys	Slight hypertrophy, atheroma of vessels
abundant	oedema	800 to 1000 grms.		Pneumonia	Kidneys	Hypertrophied
none		1005 6 to 8 pints	Tumor, supposed cancer of stomach		Kidneys	Hypertrophied
copious	blood	550 gms.		Pulmonary Oedema.	Liver & Kidneys.	Fatty, endo & peri-arteritis

	Date	Sex	Age	Name	Reporter	Previous Illness	Symptoms.
55	1881	M.	50		Pye-Smith	Drunkard	No renal symptoms, no uraemia.
56	1882	F.	35	F.	Chotinsky	Vomiting, loss of appetite, constipation, for 2 years.	Cachectic, thin.
57	1882	M.	73	D.	Brodeur		Slight dyspnoea on walking & going up stairs.
58	1882				Merklen		
59	1882	F.	56	E.D.	Babinski	Pain in abdomen 15 months ago, increase of weakness.	Loss of flesh, diarrhoea, pain in abdomen specially to left.
60	1882	F.	40		Sabourin		

Albumen	Blood & oedema	Sp.gr. etc.	Tumor	Cause of Death	Organs Cystic	State of Heart
trace		low		Chronic Pneumonia.	Liver & Kidneys	
much	no oedema, a little blood daily	Scanty	No tumor	Uraemic Coma	Kidneys	Normal
small	slight oedema	pale		Pulmonary Oedema.	Kidneys	Hypertrophied
					Ureters, bladder & kidneys	
albumen	blood	1½ litre	On left side in flank	Coma	Kidneys & liver	Large
traces				Coma	Liver & Kidneys	

	Date	Sex	Age	Name	Reporter	Previous Illnesses	Symptoms
61	1882	F.	54		Chotinsky	Sarcoma of Humerus.	Wasted.
62	1883	M.	53	R.R.	Mahomed		
63	1884	F.	48		Leichten-stern.	1½ years	Anemia, oedema of face.
64	1884				Cheron		
65	1884	M.	43	X.	Cheron		Vomiting
66	1884				Valude		Cachectic
67	1885	F.	48	Mrs S	Roberts	Sudden vomiting a week ago, convulsion.	
68	1886	M.	50	A.L.	Wagner	None	
69	1886	M.	37	H.B.	Schachmann	Pain from time to time in either flank, since age 18.	Pain in left flank, sleeplessness, loss of appetite, vomiting
70	1885	F.	63	MJT.	Caresme	Pain in right flank for a time. Phthisis.	Pain
71	1886	F.	47	M.C.	Lejars	Pain for 15 years in loins. Haematurea 2 mos ago.	Thin, pale, cachectic.
72	1886	F.	41	M.M.	Wagner	Sudden pain in abdomen 3 weeks ago, sickness, diarrhoea	Thin, apathetic
73	1887	F.	53	L.	Lejars		Lumbar pain, vomiting anuria, diarrhoea.
74	1887	M.	49	F.	Legrand	Headache & oedema for 2 years	Continual headache.

Albumen	Blood &c.	Sp.gr.	Tumor	Cause of death	Organs Cystic	State of Heart
a little	Some			Coma	Liver and kidneys	
					Kidneys & dilatn of liver ducts.	
little	Oedema	low 2-4 litres		Uraemia	Kidneys	Hypertrophied, arteries rigid
					Kidneys	No hypertrophy
				Coma	Kidneys	Hypertrophied.
		Retention		Cachexia	Kidneys	
1/12		1013 Anuria 4 days	Both flanks	Convulsions & death	Kidneys	
None				Coma	Kidneys	No Hypertrophy
Much		1200 grms		Coma	Kidneys	
			Rt flk for 8 or 10 yrs.	Phthisis	Liver, uterus kidneys.	
None			Right side	Coma	Kidneys	Hypertrophied
Traces	No Oedema	1009 1 litre		Collapse from haemorrhage 3 days after delivery.	Liver and kidneys.	Normal
					Kidneys	
None		1800 to 2000 grms		Convulsions & Coma	Kidneys	

	Date.	Sex	Age	Name	Reporter	Previous Illnesses	Symptoms
75	1887	M.	33	E.F.	Hommey		Inflammation of right hand.
76	1887	F.	88	D.	Hommey		Vomiting, slight dyspnoea.
77	1887	M	46	J.C.	Hommey	Frequent headache, vomiting and epistaxis	Thin, dark, pigmented skin
78	1887	M.		C.	Hommey		Tumor in pectoral region.
79	1887	M.	62	J.V.	Courbis	Tumor in epigastrium & right hypochondrium	
80	1888	F.	47		Prudden	Pain in abdomen for 3 months.	Oedema of thighs, pale
81	1888	F.	42	C.E.	Thiriar	Pain in stomach and Rt Flank for 9 yrs.	Recently pain worse.
82	1888	f.	45	Mrs F	Ritchie	Cachexia vomiting constipation for more than a year	Cachexia, thirst, vomiting
83	1889	M.	35		Jackson		Maniacal, 2 weeks ill
84	1889	M.	46		Newman	Pain in left side, later in right also.	Dyspepsia, headache, giddiness.
85	1890	M.	62		Paterson	Swelling of feet, dyspnoea for 2 mos.	Anasarca, anemia
86	1890	F.	51		Webster	Dull aching pain in loins.	

Albumen	Blood &c.	Sp.gr.	Tumor	Cause of Death	Organs Cystic	State of Heart
None till nr death.				Convulsions & Coma, Pyaemia	Kidneys, left only slightly	Normal
Notable quantity		1016 dark		Coma		Slightly enlarged.
	Slight haematuria.			Phthisis	Pelvis of kidney & kidneys.	
				Pleurisy	Kidneys	
None	None	Normal	In epigastrium & Rt Flk		Liver and kidneys	Hypertrophied.
None	Oedema				Kidneys	
			In Right flank	Recovery after Nephrectomy.	One Kidney	
Trace	None no oedema	low copious	In left flank 8 yrs, Rt flk some	Uraemia	Kidneys	Not examined.
				Coma	Kidneys	Hypertrophied.
Albumen	Blood		In both flanks		Kidneys	Hypertrophied
Moderate	Oedema	1010		Convulsions & death.	Liver and kidneys	
		Normal		Exhaustion from Sarcoma of bladder.	Kidneys	Normal

	Date	Sex	Age	Name	Reporter	Previous Illnesses	Symptoms	A
87	1891	F.	40	Mrs H	Kennedy	In 1886 sickness, headache 1888 twice comatose		T
88		F.	38		Von Berg-mann	Tumor 10 years	One year pain in tumor nausea loss of flesh.	T

Albumen	Blood &c.	Sp.gr.	Tumor	Cause of Death	Organs Cystic	State of Heart.
Trace		low 5 to 8 pints.	In each flank.	Coma	Liver, right ovary, kidneys.	
Traces	Quantity	Pale	In one side.	Asthenia after Laparotomy	Kidneys	Healthy.

one case which was overlooked was only 5, and Cruveilhier gives a drawing of one kidney from a child age 3, the other kidney was healthy.

In analysing these cases the results cannot be absolutely accurate seeing that the information given by various reporters is very imperfect, but the error is more likely to be on the negative than on the positive side. In 10 cases there is no history given, and in many of the others there is little detail even in relation to important points.

Age. of the 88 cases the age is given in only 75, although it is stated that two were aged. The average for the 75 is 45.3 years, the youngest was 23, and the oldest 88. *see opposite*

Sex. 37 were females, 45 males, and of 5 the sex is not reported.

Date at which the symptoms appeared.

1	22 years	before	
1	19	"	"
1	15	"	"
3	10	"	"
2	7	"	"
5	several		"
1	4	"	"
2	3	"	"
1	2 1/2	"	"

1	1 year	before	
2	9 months	"	
1	8	"	"
1	several		"
1	2 1/2		"
3	2	"	"
1	1	"	"
1	3 weeks		"

The previous symptoms were chiefly, pain in loins or abdomen, haemorrhage, gastro-intestinal disturbances, oedema.

Symptoms.

of the 78 cases, 30 had symptoms, apart from albuminuria, pointing to some renal affection, 20 complained of pain in the loins or in the abdomen, 19 had haematuria at some period of their history, 16 at the time of examination. 7 were reported

to have had no kidney trouble or no evidence of illness, the kidney condition being discovered post-mortem.

Oedema was known to have existed for a time in 7 cases, and in 4 more it was discovered on examination. In 10 cases it is stated definitely that there was no oedema.

In the urine of 33 it is noted that there was albumen.- Of these the amount is described as slight, or traces, in 19; as considerable in quantity in 11; and in 1 it appeared only near the time of death.

In 11 cases it is recorded that there was no albumen, and it is also to be noted that some of the others occurred before the time at which it became customary to examine for albumen.

The urine was usually copious, pale, and of low sp. gr.

Gastro- intestinal disorders were noted in 19 cases - vomiting, constipation, diarrhoea, and loss of appetite,-but it is evident that they occurred much more often seeing that in other 12 cases loss of flesh is recorded, without reference to gastric disturbance. 20 are stated to be anemic, cachectic or to have suffered from loss of flesh.

Dyspnoea or some pulmonary affection was present in 13 cases.

In 21 tumor was discovered during life in one or both flanks, in one of these there was tumor in the epigastrium also, and in 2 other cases in the epigastrium only. Of the 21 cases tumor was found on both sides in 8 cases - 5 times in

* Henry Morris (100) had a very exceptional experience. In two out of his three cases only one kidney was affected.

the right side only, and five times in the left only -, in 2 cases it is not indicated on which side the swelling occurred. In 2 cases it is stated definitely that there was no tumor.

Cause of death.

The cause of death is not recorded in 16 cases. Of the remaining 72 the following is an analysis.

Coma	20
Convulsions and Coma	8
Uraemia (one of these called delirium	10
Pulmonary oedema	4
Diseases of the chest	9
Cerebral Haemorrhage	8
Heart and Aortic diseases	2
Sudden death in bed	1
After operation	2
Haemorrhage after delivery	1
Pyaemia from surgical injury	1
Exhaustion	6
	72

Post Mortem.

On post mortem examination it was found in all of the cases except two that both kidneys were affected. Of the exceptions the left kidney was involved in one case, and in the other exception it is not stated whether right or left. It is noted 8 times that the left kidney was the larger, in 6 that the right was larger than the left, but in the other cases no comparison is instituted, except that once it is remarked that one kidney was large the other small.

In 21 cases the liver also was cystic. Of these it is noted in one case that the Thyroid was similarly affected, in one the uterus, and in one that the right ovary was cystic.

Of the other cases cystic disease was found in one ovary, in another case the uterus and bladder and in one the pelvis was affected with cystic disease.

The condition of the heart is stated to have been

Normal in	9	cases
Hypertrophied	29	"
Dilated	3	"
Fatty	2	"
Arteries Artheromatous	6	"

Typical Clinical History.

An analysis of these cases shews that, whereas it occasionally happens that an individual may have cystic disease of both kidneys without any prominent symptom, and that a rapid fatal termination may result from the disease, in the large majority of cases there are indications and discomforts of various kinds.

The earliest symptom noted is pain in the loins, not constant but paroxysmal, and there may be the occurrence of haematuria. At an early period the urine is not increased, but as the disease advances it becomes copious, pale, of rather low sp. gr., with or without a trace of albumen, then there may supervene various gastric, pulmonary and nervous symptoms due to the retention in the system of morbid material. Sickness and constipation are common, loss of appetite, loss of flesh, and thirst, bronchial affections, dyspnoea, headache. On physical examination, if the patient is thin a tumor may be found on one or both sides and

probably the heart is found to be hypertrophied. In this condition the patient is liable to various complications of an inflammatory nature, and while the disease may run its course to a fatal termination without either oedema or albuminuria, both of these symptoms may occur in consequence of inflammatory affections of kidneys and of chest. In very advanced cases the patient is in a very pitiable condition, much emaciated, prostration great, the thirst urgent, sleep much interrupted in consequence of frequent micturition, appetite gone, occasional retching, dyspnoea troublesome.

The fatal termination may be sudden, due to cerebral haemorrhage, or to uraemic coma. There may be more than one attack of cerebral haemorrhage. The fatal illness may be a long one either in consequence of the miserably cachectic condition which has been induced, or by reason of uraemia lasting for a considerable period and terminating in coma and death.

The clinical history has a great resemblance to that of Cirrhotic Bright's disease with certain differences. In cystic disease of the kidney pain in the back or abdomen, of a paroxysmal character is more frequent, haematuria is a not uncommon circumstance and should be looked upon with suspicion.
The heart seems to be less frequently hypertrophied than in Cirrhotic Bright's disease. When the condition of the abdominal wall permits of a satisfactory examination being accomplished, the presence of renal tumors enables us to differentiate the

two maladies.

The Microscopical Characters of the Kidneys.

These I have studied from the following specimens.

I. The large cystic kidneys from the patient whose history is recorded page 15 et seq.

2. Portion of large cystic kidney received without history.

3. Large Cystic kidneys received from a friend who found them postmortem in a woman who presented no symptoms of renal disease.

4. Portion of large cystic kidney received without history except that the liver contained many large cysts.

5. Portion of considerably enlarged kidney, a great part of which was transformed into cysts.

6. Cystic kidneys from a full grown pig.

7. Piece of cystic kidney and of cystic liver from a patient who had also waxy disease of liver, kidney, and spleen.

Unfortunately the epithelium of the specimens Case I. was partially shed because they had been placed for some hours in solution of Chloral. In case 3. the kidneys were decomposing before they were received, in them also the epithelium was shed and does not stain well.

Case I.

* Sections Fo. 40 to 46

Microscopical examination of sections of these kidneys* shews such a displacement of parts by the cysts that there is some difficulty in recognising the various elements in many regions,

or rather which portion is under examination. For convenience the usual elements of the kidney will be first examined, then the cysts.

1 Kidney structures

a) A striking feature is the very large amount of connective tissue which is nearly everywhere present (Pl.W Fig 12*). The large amount which surrounded the organs has been already noted, and it is a very marked feature of most sections, in some there is such an increase that the normal structure is almost entirely obliterated, in others a few tubes fairly healthy are seen traversing a broad fibrous band. In the neighbourhood of the larger cysts the connective tissue is usually much increased, but this is not invariably the case. In some places there is a considerable amount of fat. Evidences of inflammatory change are exhibited in many areas which are infiltrated with wandering leucocytes, and there are also many large connective tissue corpuscles. These changes are seen equally in the cortex and the medulla.

* Sections Fo 40546

b) The walls of the bloodvessels are thickened with an Ipecacuhana root appearance, many of the Malpighian tufts are enlarged, some of the capillaries - possibly newly formed ones- have delicate walls, because although very little pressure was used for the carmine injection the vessels had given way seriously damaging the specimen.

c) The Secreting Structures. The Malpighian tuft is often enlarged. The capsule, although sometimes unchanged, is generally very much thickened. Where subjected to pressure by adjacent cysts it is frequently distorted and closely adpressed to the glomerulus. It is frequently seen to be dilated, and that in different degrees, all the capsules in an area are similarly affected. The capsules alone may be dilated, or both capsules and tubes. In several of the dilated capsules there may be seen the compressed remains of a glomerulus. The tubules in the neighbourhood of cysts are distorted in consequence of compression, some have the lumen entirely obliterated, on the other hand they are frequently dilated little or much, many or all the tubules in certain areas to a similar degree (*) Pl VI Fig 1 Section 45

The membrana propria is thickened, the increase is sometimes considerable, and in a few of the sections it is constricted and rugose (**) giving the appearance of an outgrowth into the lumen of the tubes. Section 43 The epithelium is altered in a variable degree, sometimes the nucleus does not stain, or stains badly, sometimes it is shrunken. Occasionally the epithelium is atrophied from pressure, and there is frequent evidence of colloid degeneration. Many tubes contain granular material and in some areas many of them are filled with colloid matter.

2. The Cysts.

As has already been noted these are of very variable size and shape, they are as frequent in the medulla, as in the

cortex. Some are round others oval, others variously modified by pressure of adjacent cysts. Of some the outline is regular, but others have the regularity interrupted by large or small projections into the cavity. On section these have in one place the appearance of a ridge, at another they are long and finger-like. Some of these projections contain many tubes and blood-vessels, or it may be that in such a projection the remains of a single tube are to be seen. The projections have in many cases broken extremities, and are evidently portions of dissepiments which have been broken across, but frequently they are rounded and have a continuous epithelial covering (this is however better seen in sections in which the epithelium is better preserved, those sections marked 'Ro') The cyst walls are of variable thickness, they are invested by epithelium, squamous or cubical.

The contents of the cysts have been already described as to their appearance and chemical qualities. The reddish fresh fluid from one of the cysts exhibited under the microscope a small amount of Cholesterine, numerous white granular cells resembling white blood corpuscles which when treated with solution of Potash shewed 2 or 3 roundish nuclei, a few large compound granule cells, a large quantity of granular matter. The contents of another cyst, a dark fluid of thickish consistence, under the microscope shewed many round cells about the

size of red blood corpuscles, these were very thin walled with two, three or four strongly refracting round bodies, usually near the edge. A few compound granule cells, and considerable amount of yellow amorphous matter in large masses. A thickish white fluid with more solid masses, taken from another cyst, shewed a large number of round corpuscles from the size of a white blood corpuscle to 3 or 4 times that diameter, all granular on the surface, much cholesterine and granular matter.

As seen after hardening and staining, many cysts contain finely granular material, many a yellowish substance in roundish and irregular masses, probably colloid matter and this appears like little droplets adhering to the epithelial lining of the cysts.

Case II.

Has many points of resemblance to case I., specially in the large amount of coarsely fibrillated connective tissue which is almost everywhere present. It contains in some places even a larger number of wandered leucocytes than the former specimens (*), and contains many large connective-tissue corpuscles. The glomeruli have a large number of wandered cells. In the connective tissue there are very many blood-vessels in some localities, probably some of them newly formed. The walls of the arteries are thickened.

* Sections Ru. 52 & 55

The tubules are seen in some sections dilated, * in others compressed by the broad fibrous band through which they pass

* Pl. VII Fig 1.

Pl VII Fig 2 (*). Some of them are filled with colloid matter, and in some there may be seen wandered leucocytes, and deeply stained nuclei. The nuclei of the epithelium in some places stains well in others badly or not at all.

The cysts are of varied size and shape and have a lining of squamous or cubical epithelium (**).

** Pl VII Fig 2 Sections 52.53

While examining this and the former case the question arose as to whether all the cysts have an epithelial lining. In many of them a few cells could be seen adhering at intervals, but in some of them only a thick fibrous membrane or band could be seen and no epithelial investment at any part. It was a question whether the lining had been separated in process of preparing and mounting the specimens, or whether, after a time while still in the body it was shed and not replaced. After making use of the paraffin method of cutting sections, epithelium has been found in all the cysts, and if not actually in situ adhering to the wall, it is got in bands within the cavity of the cyst. *

52. 53

Case III.

Two kidneys from a woman age 51 who died of Sarcoma of the bladder, up to the time of the development of the Sarcoma she was not known to have had any urinary trouble. She died from exhaustion. On post-mortem examination the kidneys were unexpectedly found to be cystic. They were considerably enlarged, covered with cysts, and on section cysts were seen in every

part of the organs both in medulla and in cortex. They were of all sizes from that of a filbert to those which could only be distinguished by the microscope. Unfortunately the organs were decomposing when they were recieved.

Sections 47 to 51

Under the microscope the capsule is seen to be thickened, and there is a great development of finely granular connective tissue everywhere, with large nuclei. In some areas there is little else than connective tissue to be seen whereas in others the increase is not great.

The size of the glomerules is in marked contrast with the large Glomerules of the two former cases, specially of case I. The capsules are in some places dilated all in one area to nearly the same degree (*). Some of the tubes also are dilated, and in one area in which some convoluted tubules are dilating, there is seen the mode of formation of some at least of the smaller diverticula which so frequently occur in the cysts - the portion between two curves of a tube is left projecting into the dilated cavity.#

* Pl. VIII Fig 1 Sections 47. 9 48.

Section 49.

Cysts are to be seen of all sizes and although the epithelium is generally desquamated it is found in some cysts still in situ, in the others it occurs in bands or scattered about in the cavity, it is squamous or cubical.

Case IV.

Portion of a large cystic kidney, no history except that

the liver also contained many large cysts.

This kidney was in a better state of preservation than any of the other three previously examined.

a) The amount of connective tissue is considerably less than in the others, and not so generally distributed. In some areas there is such an increase that the normal structure is almost entirely obliterated(x), in other portions the tubules are not much altered. In the neighbourhood of the large cysts the connective tissue is usually much increased, but this is by no means invariably the case, around some of the smaller cysts there is no evident increase, indeed beyond the epithelium there is hardly any special wall (xx). The connective tissue occurs also at a distance from cysts, and without any evident relation to the blood vessels.

x Pl.IX. Fig 1. Section 66

xx Pl.VIII. Fig 2. Pl.XII. Fig 1. Section 59

Evidences of inflammatory action are seen in wandered leucocytes, and in increase of connective tissue corpuscles, and these changes are well marked in certain limited areas.

b) The walls of the arteries are thickened. The glomerulus seems in a few cases to be enlarged.

c) The capsule of Bowmann is occasionally found dilated with the glomerulus compressed. The capsule is not in general much changed but occasionally it is thickened. The tubules are in some places dilated. Both the Malpighian bodies and the tubules in the neighbourhood of cysts are found compressed, distorted,

† Pl. VIII. Fig 2. and their cavities obliterated (†). The membrana propria is thickened, particularly in certain districts, the increase is not nearly so great as in cases I. and II., but in a few of

†† Pl IX. Fig 2. the sections it is constricted and rugose (††), giving the appearance of an out-growth into the lumen of the tubes.

‡ Section 28 The same condition is found in cirrhosis (‡), it is not due to sections of spiral portions of tubes, the projections are too close for this, and they are too wide and too deep to be prolongations of the membrana between the epithelial cells.

In this case the epithelium is not seriously altered in the greater portion of the structure examined, the nuclei in parts shrivelled and not staining well. Many of the tubules in certain areas are filled with colloid matter.

2. The cysts in shape and size vary as in the other specimens. The diverticula are well marked, many of them contain only a few tubes in section. They have in many cases broken extremities and are evidently portions of dissepiments which have

℗ Pl. XI, Figs. 1. 2 been broken across℗, but frequently they are rounded and have a

= Pl. X, Figs. 1. 2 continuous epithelial covering (=). These occur not only in cysts of considerable size but also in comparatively small ones. Those which are broken across are most frequently seen in cysts of medium and large size. In cysts of moderate size complete partitions are frequently met with, some of them extremely thin consisting of a double row of epithelium with

‖ Pl. XI, Fig 1 Sections 62+63 almost no inter-cellular substance‖. Inthese partitions tubes

or their vestiges are seen, even in the very attenuated dissepiments the transverse sections of one or more tubes at intervals is a prominent feature (*). Pls. X. XI Sections 62 & 63

The cyst walls are of variable thickness having blood-vessels ramifying in them. Frequently the cyst wall is surrounded by a dense layer of tubes and Malpighian bodies colsely packed and adpressed, ** in other cases a patent tube or blood vessel presents a semicircular elevation of the cyst wall and bulging into its cavity. The cyst walls are invested with epithelium squamous, cubical, or columnar (#), the last is not so often found as the two former. In this respect this affection of the kidney is an exception to the rule in cystic diseases, but it agrees with these in the variety of character of epithelium which occurs in the same cyst. The kind of epithelium seems to have no relation to the size of cyst except that columnar is more comm in the smaller than in the larger cysts, in the latter squamous is common, but squamous is frequently found in the very small ones. In some of those with cubical and columnar epithelium there is evidently an active proliferation giving on (ℙ), the appearances shewn are not confined to one or two sections, but can be traced through many of a series, shewing that the piled up epithelium is not an appearance due to the section having been made obliquely through the investment of an adjacent cyst wall which made a somewhat acute angle with

Pl. VIII, Fig 2.

Pls. X. XI. XII. Sections 64. 38

ℙ Pls. X. XI, Figs. 2 Sections 38 60. 61. 62

the one which is cut transversely. In those cysts of which the epithelium is an active state of proliferation there are found free cells staining deeply, and sometimes compound granule cells.

Case V.

Portion of considerably enlarged kidney, a great part of which was transformed into cysts, some of them with solid or semisolid contents. This kidney has hardly the appearance of the large cystic kidney, it certainly is larger than normal, and has undergone very considerable cystic transformation. The portion which I recieved for examination was the only part which seemed not to have undergone great cystic change.

Sections 69 & 73 Under the microscope it is seen that over considerable areas there is not much change in the intertubular stroma, but in other portions the increase is evident. Bands of hyaline connective tissue, with well marked nuclei, radiating in different directions, and specially seen between and connecting the Malpighian bodies. In limited areas there are wandered leucocytes. In some of the new bands there are tubules distorted and atrophied, the epithelium having undergone absorbtive and colloid changes.

The capsules of the Malpighian bodies are generally thickened some of the glomeruli are seen undergoing colloid change. The lumen of the tubules is rarely altered, a few of them are

filled with colloid material, but the epithelium over extensive areas is altered, with the nuclei absent or hardly stained, and scattered about in these areas are tubes which are much more healthy.

The cysts have very thin walls, in some of them the wall can hardly be distinguished beneath the epithelial investment. The epithelium is generally cubical but in some squamous. In some of the cysts the wall has been torn in the process of preparation, and sections of tubes are seen within the cyst in the neighbourhood of the tear.[×] Sections 72. & 73.

As I have already pointed out the Malpighian bodies are very often seen in pairs, or three or four together, they are developed in pairs ([××] Sections 17 to 27), and in sections of this kidney, just as we have observed in cirrhosis, occasionally the second of a pair is dilated, filled with granular or colloid matter, lined by squamous epithelium, and is evidently undergoing cystic change.[#] Sections 72 & 73.

In these sections the peculiarities are, the small amount of connective tissue around the cyst wall, the evidence of the development of cysts from Malpighian bodies, the small amount of change generally in the contour of the tubules, and the evidence of the newly formed tissue causing constriction of secreting structures.

Case VI.

Cystic kidneys from full grown pig, no clinical history.

Each kidney formed a slightly elongated rounded mass, its surface smooth and regular, mostly pale, but with reddish patches scattered over it.

After section in the ordinary way, the capsule could be readily removed, it was not adherent, the wall of the pelvis was much thickened, with firm bands passing out to the cortex. The whole of the interior of the organ appeared to consist of cysts of different sizes (× *Pl. XIII.), not imbedded in much stroma, but rather moveable, and without much difficulty individual cysts could be dissected out and removed entire. The cysts were grouped into bundles corresponding to the cones, when these groups were drawn apart from each other, prolongations from the thickened wall of the pelvis were seen to pass outwards, apparently to the capsule. The small reddish patches which were seen on the surface of the kidney were found to be from an eighth to a sixteenth of an inch in thickness, and on further examination these were found to be the remains of the kidney secreting substance. The appearances indicate that the cones had been almost entirely transformed into cysts, that the cortical part was compressed under the capsule, and that the prolongations of the pelvis forming the calyces had been stretched and carried quite to the periphery of the organ along with the compressed cortical

substance. No kidney structure could be discovered between the cysts. There were no cysts within cysts, although some of the smaller ones bulged into their larger neighbours.

The fluid removed from the cysts was yellowish, with only a very slight flocculent deposit, sp. gr. 1010. albumen 2. 3 o/o as determined by means of Esbach's tubes, Urea 0. 3 o/o, chlorides abundant. No hooklets could be found.

Several portions were removed from the reddish islands under the capsule they were hardened in spirit and prepared by the paraffin method.

Sections 74. to 78.

On microscopical examination it is found that a portion of the structure removed, varying in depth from one to three sixteenths of an inch, is less changed than the other and deeper part which is most removed from the capsule and which consists of cysts with much fibrous change in their walls. Here and there in patches there is a considerable infiltration of leucocytes, and an increase of the connective tissue nuclei as well as of the intertubular stroma (** 74 to 78). The Malpighian capsule is occasionally slightly dilated and contains granular material, in some the nuclei of the epithelium which lines the capsule seem to be unduly crowded together. Many of the tubules have the nuclei shrivelled, or absent or very few in number. The deeper part consists mainly of cysts. In the cyst walls there is much connective tissue some of it fibrous and some hyaline looking. In these walls are urinary tubules (# Pl XIV Fig 1) in various degrees

of dilatation, or of compression and distortion. The cysts are lined by epithelium mostly cubical with large roundish or oval nuclei, and in some places the epithelium is piled up in several layers.[+] Some of the cysts have squamous epithelium. There are diverticula in several of the cysts with a complete epithelial investment, and some of these projections contain remains of tubes. Although here and there in the cortical substance there is an increase of fibrous tissue, it does not at all resemble the broad fibrous bands commonly seen in chronic interstitial nephritis as it occurs in the pig ([xx]).

[+] Pl. XIV. Fig 2

[xx] Section 35

Case VII.

Piece of cystic kidney, and of cystic liver, from a patient who had also waxy disease of liver, kidney and spleen.

The kidneys were not much enlarged but they were full of small cysts scattered about both in the medulla and in the cortex.

Microscopic examination of the liver reveals a marked increase of connective tissue in the portal spaces, with many leucocytes, and increase of connective tissue corpuscles. The intra-lobular veins are in many places much distended.[xx] The liver cells chiefly about the centres of the lobules are widely apart ([P]), and in some places atrophied.[O] These changes are in marked contrast to the appearances in a liver supposed to be healthy ([#]). Many of the cells

[xx] Pl. XVI, Fig. 1. Section 81.

[P] Pls XV. XVIII, Fig 1.

[O] Pl. XVII, Fig. 2. Section 81.

[#] Section 82.

chiefly in the intermediate and outer zones are waxy. The liver cells along the margins of the veins are in some places arranged in lines * and some of them atrophied, perhaps it is this condition which led Juhel-Renoy to note that he had seen the change of liver cells into epithelium of new bile ducts.

* Section 83

I have not been able to trace the increase of bile ducts noted by Mallasez. The cysts have a fibrous wall with many connective tissue corpuscles, and sometimes leucocytes. The cyst wall has a complete lining of cubical epithelium with large oval nuclei, corresponding exactly with that of the smaller bile ducts. In some of the cysts there is to be seen an accumulation of epithelial cells at certain parts of the walls (*), in others there are small projections from the cyst into the adjacent connective tissue, these have the appearance of, and give the impression of a process of budding from the larger cyst (**). Around it there are larger pouches, and also what seem to be small cysts, but what may be only sections of other pouches. These appearances have been observed by Naunyn who believes that the cysts grow by budding of the epithelium, and he classes them with Adenomata.

* Pl. XVI, Fig 2 XVIII Figs 1.2. Sections 79. 80

** Pl. XVIII Fig 2 Sections 80

In the kidneys of this case there were found many small cysts scattered through both cortex and medulla. On microscopic examination although there is not a great general increase of the intertubular connective tissue, in some places

the increase is considerable and in many others the nuclei seem to be particularly large and numerous, a few limited areas are infiltrated with leucocytes.

Some of the bloodvessels are waxy, some of the glomerulihave undergone a waxy and some colloid change.

Of the tubuli many have the epithelium swollen, and the nuclei absent or few in number, adjacent tubes seem to be quite healthy. Many of the tubes are filled with colloid looking matter.

The cysts are of various size and shape. Many of them have very thin walls, lined chiefly with cubical epithelium, in a few the investment is squamous, in a few cylindrical, but in most it is cubical. It is to be noted that those with cylindrical epithelium have among the cell contents scattered cells and nuclei which have stained in various degree (*). The thinness of the cyst wall is a striking feature in some of the sections, dilated tubules are seen projecting into the cavity (**). In this kidney the cysts are never found of very large size and probably in consequence of this there is less pressure on the adjacent renal tissue, consequently the tubules adjacent to cysts are not nearly so much distorted as in the very large cystic kidneys.

* Pl. XIX, Fig 2 Sections 85 & 86

** Sections 85

In some of the sections, when examining with the low power, the eye is attracted by some of the cysts and tubules

which have the investing epithelium closely crowded, and the large nuclei deeply stained (*). Under the high power cysts with this character are seen to have small recesses, and also papilli-form projections, all lined or surrounded by closely packed cubical epithelium with large oval nuclei, and in the adjacent stroma similar nuclei are seen packed together, and from the cysts there are small projections all of which give the idea of a budding process. The appearances are almost identical with those seen in the liver.

*Pl XIX Figs 1.2

Summary of microscopical appearances of all specimens.

In all of these specimens we discover certain features possessed by all of them , and certain points of difference.

The amount of connective tissue is very varied, in all the specimens it is increased, and in the most advanced cases of the disease the increase is very great. It is most marked as a rule in the neighbourhood of the cysts, but this is not invariably the case, some cysts of considerable size beingsurrounded by little newly formed tissue, and some of the smaller ones having much, it also occurs at a distance from cysts without evident cause either in relation to cyst or to blood supply. In the advanced cases it is very fibrous, in some of the earlier ones more hyaline. In all of them there are evidences of local irritation in the presence of leucocytes and increase of connective tissue corpuscles, and although these are more marked in the advanced than in the earlier cases, they occur in both at a distance from cysts.

The thickening of the coats of the arteries is more or less marked in all the specimens.

The enlargement of the glomeruli in the advanced cases is a striking condition.

In all the cases there are changes in the tubules and Malpighian bodies, in most of them there are to be seen all stages from simple dilatatiom to cyst formation, in most of them the remains of a glomerulus in one or more of the capsules may be seen, also the degeneration both of glomeruli and of epithelium. The changes in epithelium and the nuclei are common to all in varying degree, in some the nuclei are shrivelled or absent over considerable areas, and in all they stain badly in districts of considerable extent.

The cysts have nearly the same characters in all as to size, shape, projections into their cavities, epithelium and surroundings, but in the advanced cases the distortion and distruction of surrounding urinary secreting structures are more marked than in the earlier ones, and in the latter the proliferative changes are much more abundant than in the others. This is specially evident in the specimens marked"Ro" and"Wi", in many of the cysts in these the proliferation is marked by a piling up of epithelium, by the multiform character of it, by the cyst contents including cells and nuclei which take the stain, and the specimens"Wi", which are the least advanced, shew a process of budding. In cystic disease of other organs I understand

that it is chiefly in the earlier stages that the budding process is seen, and when the cysts have attained some size it can be no longer traced. Mr Stiles tells me that this is his experience as regards cystic disease of the mamma.

The cyst contents, so far as examined, correspond with each other in giving evidence of the elements of the urinary secretion.

The specimens marked "Pg" differ from all the others in so far that the disease has affected extensive tracts of the papillary region, and the cortical substance in connection with them, and in the rest of the cortical part which has been examined no cysts occur.

Historical Sketch of the Literature of the Subject.

Boyer (1) in 1822 described a case of the disease in connection with the clinical history. This is the first of which I find record.

Bouillaud in 1827 (2) wrote on urinary infiltration and cysts of the kidney, and states that the affection ahich he designates by this name has not to his knowledge been described by any pathologist. Although three of the cases which he records may have been general cystic degeneration of the kidney there is not sufficient information given to differentiate them from contracting kidney with numerous cysts.

he describes two sources of formation viz. the cellular tissue of the kidney, also a capsule of which the communication with the tubule has been obliterated.

He says, as this disease occurs at an age at which paralysis is common, and there is at least a notable feebleness of the excretory organs of the urine, he is led to believe that the malady may have some relation to an obstacle to the flow of that liquid, that the urine will distend the substance which secretes it, and the tissue losing its resistance may permit the liquid to filter into the parenchyma, and to deposit itself in the connective tissue which surrounds it.

Rayer (3) in 1841 was the first to give a careful description of the anatomy of the disease, he records several cases, and recognises that, without appreciable antecedent renal affection, the two kidneys are affected by a true cystic degeneration of the cortical substance, a degeneration which may reach such a degree that there remains almost no trace of renal substance. Then the urinary functions are arrested or perverted, and grave functional lesions of other systems, above all of the cerebro-spinal, determine death. Complete cystic degeneration is rare, and has been more observed by pathologists than by physicians.

Cruveilhier (97) has depicted in his atlas a case under the title of Cystic transformation of the Kidney, *see opposite*

Hughes Bennett (13) states that cystic kidney may originate in 3 ways

1) Tubular obstruction of various kinds viz. coagulated exudation, pus, blood, altered epithelium, different salts.

These cysts are of different sizes and their contents various.

2) Cysts around glomeruli, would appear to arise from obstruction at the commencement of the duct. The cyst seldom exceeds a small pea. The tuft is pressed upon and atrophied.

3) From the cells.

Simon(5)1847 held that certain diseases of the kidney (whereof subacute inflammation is by far the most frequent) tend to produce a blocking of the tubes, this obstruction directly or indirectly produces rupture of the limitary membrane, and what should have been the intratubular cell growth continues with certain modifications as a parenchytic development ..... These cells are organized for secretion into their cavities so as to at least draw from the blood, if they cannot eliminate from the body, the materials which fill them.

Gairdner (6) 1848 from the occasional appearance of alternate distension and constriction presented by the tubes when undergoing obliteration, is induced to believe that these cysts may be formed by the occlusion and isolation of portions of tube which have not yet lost their power of secretion. The larger cysts in the kidney present very strong evidence of being formed in connexion with the secreting membrane.

Coote (8) 1851, divides renal cysts into 4 varieties. The large cystic kidney he believes to be due to dilation of Malpighian capsules, and he supposes that the obstruction is

due to blood, tubercle, or fatty change in the epithelium. Quekett examined the organs in this case.

Lebert (10) believed that the cysts both of liver and kidney have their origin in the glandular canals.

Abeille (9) 1852 held that they arise in the cellular substance which surrounds the renal vessels, and that they are a true cystic degeneration.

Beckmann (16) 1856 believed that they may arise from the connective tissue corpuscles, but that they also result from the strangulation of uriniferous tubules, and Malpighian bodies for he found the remains of glomeruli in cysts.

Hemsbach (17) considers that the cysts arise out of the smallest nuclei which occasionally one sees scattered in great number between the urinary tubes, and which form themselves into mother cells and then receive a cellular tissue investment and an internal epithelial lining.

Goodfellow (21) 1860 states that cysts are almost a necessary and inevitable, and not simply an incidental part of the changes which constitute one or more of the forms of Bright's disease. He seems to hold that they are formed in three ways. 1) That described by Dr Geo.Johnson, that in all forms of Bright's disease, and from causes in the different forms, the tubes become obstructed and form cysts filled with a fluid not generally identical in composition with urine but resembling the fluid contained in cysts found in other parts of the body.

2) That suggested by Mr Simon, an abnormal development and growth, or an abnormal growth of the normal epithelial cells. Mr Simon states that the nutrition becomes altered, leading to an abnormal development of the germs.

3) By spaces, formed in the fibrous structures, which become filled with fluid.

Virchow (24) 1863 states that in cystic disease of the adult the problem is more complicated than in the foetal disease. He thinks it possible that a partial foetal degeneration may persist until an advanced period of life. But the ordinary course of development consists rather in the fact that cystic kidney of adults has no relation to the retention of urine, but that they develop in consequence of interstitial nephritis which is related to the excretion of solid albuminates in the interior of the uriniferous canals, these are dilated by the albuminates, and transformed into a series of varicose swellings the one behind the other.

Harley (25) believed that cystic disease of the kidney is due to blocking up of some uriniferous tubes, and consequent dilatation of the Malpighian body, which ultimately assumes the appearance of a cyst.

E richsen (26) 1864 lays the beginning of the process in the gelatinous degeneration of the epithelium.

Klein (27) 1866 observed the formation of cysts from

Malpighian corpuscles. The large cysts he believed to be formed by the union of several small ones. He traced every stage from the slightest dilation to the formation of large cysts.

Lancereaux (06) notes cystic changes in other organs concomitant with cystic kidney. He thinks that there is great analogy between the small cysts in interstitial nephritis and those in cystic kidney, in both cases the Malpighian bodies and canaliculi would be the point of departure of these numerous cysts. The epithelium would be infiltrated by colloid material which would destroy it, while the capsules and tubules would dilate, the walls would secrete a serous liquid, and blood exudation would give color. He places the origin of the process in epithelial change without discussing the influence of interstitial nephritis.

Johnson (35) 1873 states that the cysts are dilated tubes obstructed by accumulation and impaction of epithelial and fibrinous debris.

Rindfleisch (36) notes the frequent concomitance of cystic liver and kidney, and says that, with the exception of cystoid degeneration of foetal kidneys, cysts occur as complications of inflammatory changes, that they are due to strangulation of the tubes, and that they occur in the cortex of those kidneys in which an inflammatory overgrowth of the connective

tissue occurred chiefly about the larger renal vessels at the cortico-medullary junction. He considers the liver cysts to be retention cysts, in consequence of obstruction arising at any part of the biliary canal, he finds a progressive enlargement in the interlobular canals. At the commencement of the process it is always easy to find the elements of the bile.

Lecorché (37) 1875 states that they have their seat of origin in the convoluted tubules, sometimes in the Malpighian capsules.

Paget (39) 1876 teaches that most of the appearances may be explained on the supposition that the uriniferous tubules have become locally obstructed and dilated into cysts above or between the points of obstruction.

Michalowicz (44) discusses the question, he believes that malarial poison is the exciting cause, but he offers no opinion as to the anatomical pathogenesis.

Laveran (40) holds that although there may be evidence of interstitial nephritis in the neighbourhood of the cysts, at a distance the traces of inflammation disappear. That cystic degeneration of the adult is distinct from congenital cystic disease and that it is independent of interstitial nephritis.

Komorowski (41) found it impossible to say whether there were new formations or whether the cysts proceded from dilated ducts.

Greenfield (98) says that it is at present disputed whether the primary cystic degeneration develops in the same way as the scattered cysts in interstitial nephritis. Two forms of the primary disease are met with - one congenital, the other occurring in adult life. In the congenital form the cysts appear to be produced by abnormal development of renal tubules and the glomeruli. The form which occurs in adult life is rarely met with before middle age, and most commonly is not fatal until a somewhat advanced period of life. There is no doubt that the cysts result from an alteration either in the Malpighian bodies, or the renal tubules; but the primary cause and the starting point of the morbid change are at present obscure.

Grainger Stewart (99) says that nothing is known as to the causes of cystic degeneration. It may be congenital, or may come on during adult life. The mode of origin of the cysts appears to be from the dilatation above obstructed points in the course of uriniferous tubules, or at their points of origin in the Malpighian bodies. Sometimes the cysts are new formations in connection with epithelium, and sometimes they arise from the fibrous stroma of the organ.

Henry Morris (100) says that the large cystic kidney is the result of a degenerative process. The medullary and cortical portions are alike replaced by cysts. They probably owe their origin to expansion of parts of the uriniferous tubules, and atrophy of the interstitial tissue. Two of his three cases were unilateral, a very marked exception to the rule.

Ebstein (43) 1877 thought that it was not yet demonstrated whether the development of these cysts was connected with any congenital condition.

Dickinson (45) found in some cysts, four or five times the size of the Malpighian body, tubes detached from their surroundings and either little altered or crumpled and denuded. These appearances suggested to him a local destruction of tissue rather than a mere distension of any structure. He believed that some at least are attended by destruction of tissue, but that the majority are the result of transformation. He saw nothing to justify a view that they are excessive overgrowths of epithelium. He thought that it could not be doubted that cysts are as a rule altered tubes.

Malassez (47) recognised the disease to be not rare, and often accompanied by cysts of the liver. He found the mode of formation of cysts in the kidney to be difficult to study but more easy in the liver. In that organ there is first slight perilobular cirrhosis, and in the enlarged spaces one noticed that the biliary canals are more numerous than in the normal condition, very unequal in diameter, and always invested by a continuous layer of cylindrical epithelium. Some of these canals are extremely dilated and these seemed to be the origin of cysts. It is not certain that these cysts arise in pre-existing canals, it is possible that they may be the result of an en-

tirely new formation. He found variety in the form of epithelium and thought that this polymorphism lends support to the idea of mucoid epithelium.

Courbis (46) 1877 holds that the cysts in the liver arise from the formation of new ducts, and concludes that cystic degeneration both of liver and kidney accompanies or follows sclerosis of these organs, and is dependent upon an irritative lesion. That they have for seat the glandular canals, and for origin - at least in the liver - an increased formation of epithelium in new bile ducts. He objects to the name given by Naunyn, viz. "Adenoma" and would call the condition "Epithelioma", were it not that, in consequence of the meaning already associated with that word, confusion might be introduced. He holds that true cysts originate in a pre-existing epithelium, and that they have a special embryonic origin, arising from the internal or external blastodermic layer.

Roberts (48) 1879 says that cystic degeneration in the adult probably arises in the same way as congenital cases, that is by the progressive occlusion of the ducts of pyramids, leading to dilatation of urinary tubules at intervals, also of the Malpighian bodies, and finally to the formation of myriads of cysts. It is a question whether this affection is not merely a further development of a slight degree of the congenital form which has gradually increased after birth, it may be so

in some cases, but in others it seems to have commenced in extra-uterine life, and has been attributed to inflammation of the straight tubes in the pyramids, with obliteration at intervals. It is probable that the epithelium of the tubes also undergoes some active change, but of what nature seems uncertain.

Chambard (50) 1879 agreed with Malassez as to the liver, and believed that the cause was the same both in kidney and in liver.

Axel Key (49) considered the change to be congenital.

Brigidi and Severi (54) believe that there exists a formative irritation of the epithelial cells of the uriniferous tubules, and a consecutive degeneration of the newly formed cells, that the neoplasia of connective tissue is secondary to that of epithelium. They admit three classes of cysts.

1) Retention cysts, in consequence of mechanical obstruction.

2) Cysts from primary degeneration of the epithelium of the tubes - found in colloid cysts - frequent in interstitial nephritis.

3) Cysts from epithelial new formation and secondary degeneration, such as has long been admitted in ovarian cystoma.

Eve (51) 1880 found all stages of transition from dilation of tubuli to the formation of cyst like cavities. He found no evidence of the cause of retention.

Saundby (53) 1880 believes that some cysts are due to the formation of a myxomatous or gelatinous tissue from the

young cells filling the lumen, by which the basement membrane is distended and a cyst formed. He describes a similar condition in the Malpighian bodies, with or without dilatation into a cyst.

Juhel - Renoy (57) 1881 found at the level of the cortical region a development of connective tissue which compresses the tubuli contorti, these he found to be nearly all affected. In the liver he found a formation of new canals, and thought that he could trace the development of hepatic cells into epithelium of biliary canaliculi.

Pye - Smith (56) thought the weight of evidence as well as of authority to be in favour of these cysts being retention cysts, produced by constriction of the efferent bodies and consequent dilatation of either the Malpighian capsules or the cortical tubes.

Cornil and Brault (58) 1881 state their opinion that a cyst in process of formation is less a cyst than a centre of softening, that it has no proper wall, at least continuous, that the surrounding tissue does not present any indication of compression, but is transformed by a breaking down in consequence of colloid or fatty degeneration, accompanied or not by subinflammatory changes of epithelium. They state that they found in cysts not only traces of a glomerule, but several of them, and fragments of uriniferous tubules still covered by

their proper wall, facts which they could not account for by the theory of simple dilatation of a capsule or tube.

Sabourin (59) 1882 says that if these cysts are due to retention and dilatation there must be some further cause than simple retention, because these obstructions are frequent in cirrhosis of the kidney, yet cystic degeneration is rare. He notes the frequent occurrence in cystic kidneys of small epithelial tumors under the capsule - a piling up of cubical epithelium. He found side by side two cysts one with cubical the other squamous epithelium, and asks are these two segments of the same tube isolated and vegetating each in its own fashion, or two segments of different tubes. He leans to the first view, and believes that they are the result of anomalies in the nutritive evolution of glandular epithelium under the influence of general inflammatory processes of the organ. He holds that cystic degeneration is one of the accidents in the course of Cirrhotic Bright's disease, that they have as origin a special evolution of the uriniferous tubules of which the epithelium has returned to the indifferent condition, that this epithelial evolution occurs in small foci, in which the affected tubes transform themselves into alveoli with special characteristics, and that this epithelial process, occurring in the tubes transformed by cirrhosis, has bonds of relationship of the closest nature with another epithelial process resulting in the formation of adenomata. They are veritable cystic adenomata.

Kelsch and Kiener (60) think that the diverse conditions under which cysts are produced lead to the belief that the pathogenic conditions are multiple. Kidneys attacked by pure sclerosis do not contain cysts, in order that these may be produced there must be also fatty or colloid degeneration. The fatty degeneration of epithelium is found to the exclusion of sclerosis in a number of cystic kidneys. Sclerosis does not exist in the neighbourhood of cysts in process of formation, it is only a secondary development during the period of regression of small cysts, probably from irritation or compression of the surrounding parenchyma. They consider renal cysts to be centres of degeneration, related pathologically to fatty degeneration, and to a colloid fatty change in the renal parenchyma.

Green (61) 1882 believes that first the interstitial as well as the epithelial tissue of the tubules breaks down with inflammatory or medullary corpuscles, and then an embryonic tissue is produced, sometimes occupying large territories of both cortex and pyramids. Next a transformation of the new medullary tissue into myxomatous substance marked by the presence of a light, nearly homogeneous basis substance, traversed by nucleated bioplasm strings. These delicate strings perish, and a cavity is formed filled with an albuminous liquid.

C. J. Bond and B. C. A. Windle (62) 1883 found nothing to support the idea that cysts are due to an abnormal development of epithelial cells, they do not believe in the persistence

of a congenital condition, they do not think that such extensive degeneration could be produced by occlusion of tubes by coagulated blood, seeing that no haematuria occurred in their case, till a late period of its history. They suggest as explanation the formation of intertubular fibrous tissue, its contraction, strangulation of the tubes in parts, and their subsequent dilatation. They believe cirrhotic kidney to be a part of a general disease, whilst the cystic kidney is purely a local disease, not necessarily connected with changes in other parts of the body, and that the unaltered heart supports this view.

Weichselbaum and Greenish (63) 1883, consider it to be a transformation of adenoma , arising by proliferation of the epithelial cells of the tubules, with a consecutive fatty or colloid degeneration.

Bateman (68) 1884, believes that the epithelium of the kidney, as a result of some abnormal irritation acting in the adult through the blood, in the foetus through some error of development, acquires a tendency to proliferation. Whether this proliferation takes place within the tubules -as in cystic disease of the testicle -, or whether it arises in the same way as adenomata of the ovary, by an involution of the epithelial lining, the shutting off of this involuted portion from its original source, and its subsequent development into a cyst, it will be difficult to determine. This proliferation of cells

seems to spread mainly in a circular manner, the central cells being separated from their blood supply undergo colloid degeneration, the cells at the periphery multiply and so cause a gradual enlargement of what is now a cyst. This cyst by pressing on or irritating neighbouring structures becomes invested with a layer of connective tissue. The increase of connective tissue is in proportion to the size of cysts.

Goodhart (69) 1884, believes that the change is not congenital, that there is an overgrowth of epithelial cells of the tubes, which overgrowth softens in the centre, and so forms cysts. He had come to the conclusion that cystic kidney was of adenomatous nature, and formed on the principle of many ovarian cysts. He thought that cystic kidneys should be examined in that light.

Shattock (73) 1886, thought that the cysts were due to retention. All transitions could be traced from long dilated passages to the more defined circumscribed cysts. He thought that they originated in the mal-development of the histological elements of the kidney, and that the spaces are retention cysts, originating in the tubules of the Wolffian body.

Fagge (74) 1886, believed that they were formed out of the tubes of the cortex, that the affection is a form of Bright's disease, notwithstanding the one important clinical difference which exists between it and all the other forms, viz,

that the organs are often so enlarged as to be felt during life.

Rosenstein (75) 1886, saw the development of cysts out of Malpighian bodies which had been the subject of haemorrhage from the glomerulus, and also traced the stages of development from urinary tubules in the medulla.

Hommey (76) 1887, considers that the sclerosis is caused by the cysts interfering by compression with the arterial supply of the parts. He holds that the point of departure of cystic formation is to be found in the epithelium of the tubes, and that in this respect the epithelium of the kidney resembles all investing epithelium.

Bristowe (18+77) 1887, thinks that there is probably no essential difference between the cystic kidney and the granular kidney.

Duguet ( ) 1888, said that the formation of cysts depends absolutely on fibrous proliferation. They are a result of this process. Every patient who has cystic degeneration shews also sclerotic changes. Observations shew sclerosis affecting several organs, liver, kidney, breast. We cannot therefore say that there are several cystic diseases of liver, breast, testicle, etc, but that there exists in certain persons, with fibrous diathesis, a tendency to cystic transformation of glandular organs, which are the seat of proliferation of connective tissue.

Wilks and Moxon (81) 1889, hold that granular kidneys are all formed organs, that large cystic kidneys are only a variety of the condition found in granular kidneys. It is a question whether the large cystic kidney represents a lower degree of foetal cystic kidney.

Le Dentu (84) 1889, gives a very good resume but does not venture an opinion as to cause.

Le jars (86) 1889, did not find any evidence of progressive dilatation, no stages between healthy tubes and well developed cysts. He believes that for cyst formation there is something more required than occlusion of the duct or tubule, that epithelial activity must be induced. He agrees with Malassez that the disease is Mucoid Epithelioma, similar in character to that which has been demonstrated in relation to ovary and testicle. Apart from Sarcoma and Carcinoma he divides cysts of the kidney into three classes.

1) Large Polycystic Kidney, which he considers to be Mucoid Epithelioma.

2) Small cysts of Interstitial Nephritis.

3) Large isolated cysts.

Paterson (87) 1890, believes that renal cysts arise from plugging or strangulation of urinary tubules by interstitial fibrous tissue.

Kennedy (89) 1891, thinks that the disease originates in the medulla, and that the interstitial nephritis in the cortex is secondary, and more recent than the old fibrous walls and tissue in the medulla.

Summary of literature.

From the foregoing outline it is evident that, as regards the etiology of the disease, there is great diversity of opinion, a number of observers however hold somewhat similar views, and others agree in certain regards only.
We may attempt to summerize these views as follows.

Virchow, Roberts, Courbis, Axel Key, Bateman, Shattock, Wilks and Moxon give support more or less strongly to the theory of the possible persistence of an embryonic condition. Shattock is the most definite in his statements, that there are retained remains of the Wolffian body - that there is a combination of mesonephrous with the metanephros. Wilks and Moxon hold that granular kidneys are ill formed organs, and that the large cystic kidney is only a variety of the condition found in cirrhosis of the organ. Ebstein, Bond and Windle, and Goodhart express disbelief in the congenital theory.

Hemsbach believed it to arise out of the smallest nuclei lying between the tubules, that these form themselves into mother cells.

A considerable number of observers consider that the condition is consequent on cirrhosis of the kidney. Goodfellow, Fagge, Bristowe, Wilks and Moxon take this view. Kennedy considers that it is due to interstitial nephritis occurring first and chiefly in the medulla.

Beckmann, Rindfleisch, Jubel-Renoy, Paterson, and Harley believe that it occurs in cirrhosis in consequence of strangulation of the tubes by the fibrous tissue, Harley holding that it is the Malpighian bodies which are converted into cysts. Klein also has traced their origin to these bodies.

Coote and Quekett hold that each cyst commences in a single Malpighian body, and that the orifice is obliterated by blood, tubercle, or fatty epithelium. Others, connecting it with cirrhosis, state that it is induced by exudation into the tubes. Vir chow calls the exudation solid albuminates, Paget and Lancereaux call it colloid, Johnson epithelial and fibrous. Roberts holds that it is due to strangulation of the tubes along with epithelial change, and Sabourin adds to this that it is related to adenomata.

Laveran believes that it is independent of interstitial nephritis. Bond and Windle that it is a local disease with the formation of interstitial tissue and strangulation of tubes. Duguet that it is due to fibrous proliferation.

Gairdner, Paget, Lecorché, Lancereaux, Dickinson, Greenfield. Pye-Smith, Grainger-Stewart, Morris, and Eve account for it by occlusion and isolation of

portions of tubes which have not yet lost their secreting power. Other two authors also blame the blocking of tubes, viz, Simon who states that there is rupture consequent on the blocking, and a parenchytic development of tubal epithelium, while Bouillaud believes that the secretion filters into the surrounding tissue.

Abeille considers it to be a true cystic degeneration arising in the cellular substance.

Hommey, Lancereaux, and Lebert say that the point of departure is in the epithelium of the tubules. Bateman, Goodhart, and Brigidi and Severi also place the cause in the epithelium and class the disease with the adenomata. Weichselbaum and Greenish think that is a fatty or colloid degeneration of Adenoma; Erichsen, and Saundby that it is a gelatinous degeneration of the epithelium; while Lejars, Malassez, and Chambard call it Mucoid Epithelioma.

Dickinson, Cornil and Brault, and Green say that there is a local destruction of tissue, and that the cysts are centres of softening. Kiener and Kelsch also state that there are centres of degeneration related pathlolgically to fatty, or to colloid fatty change.

Critical Review of these Theories.

The first of these theories of the cause of this disease, is that which ascribes it to included remains of embryonic life - persistent germinal rudiments. This theory is a most attract-

ive one, specially if by means of it we might be enabled to bring all tumor, at least all cystic tumor formations into one category. It is true that there is evidence of such untransformed rudiments in the islands of cartilage near the epiphysial portions of long bones, specially in those cases in which ossification is imperfectly performed, viz, in rickets. It is also true that osseous tumors are very common at the external meatus, a situation at which there are so many centres of ossification that it is easy to understand how included islands of untransformed cartilage might arise. Shattock has been definite in his statement regarding the foetal elements concerned. He believes that there is a combination of mesonephros and metanephros, and that cystic kidney is due to a late development of included cells of the mesonephros. But a similar disease occure in the liver - cystic liver - an organ in which it is highly improbable that there should be any included cells of the mesonephros. In order to include the liver we would require to extend the theory, beyond the mesonephros, to embryonic elements of the epi - or hypoblast. It must be acknowledged that the epithelial investment of early foetal kidney, and the lining both of liver and kidney cysts have a strong resemblance to each other( ).

Sections 15+24 etc

We have already seen that in many of the cysts of small or medium size, and even in some of the larger ones, there are diverticula projecting into the cyst cavity and in these diverti-

cula there are renal elements - tubes or bloodvessels. The question arises, whether the existence of these is possible on the theory of cystic growth from persistent germinal rudiments. If cysts arose in the way we should expect to find them growing in the direction of least resistance, and probably assuming a roundish or oval form modified by pressure of adjacent structures, unless indeed we admit that by the process of budding, which we have seen to exist, we were to have two buds, at a little distance from each other, burrowing in the same direction, including between them some tubes, or other renal elements, which should afterwards be left projecting into the cavity of the cyst. This is possible, but I think that a more probable explanation of their origin is, that they are portions of renal tissue which have existed between the convolutions of a urinary tubule which has undergone cystic change. That this is a possible explanation of these diverticula is seen in No 49 , where a partially dilated convoluted tubule is seen in section, having projecting into it such diverticula. It may be said that in the cysts these projections are only the remains of dissepiments which have been left after absorption of the central portions. But these projections are found with a complete epithelial investment* without irregularity or break at their free extremity, in the cyst there is no corresponding portion at the other side, and they occur in cysts of small and medium size in which we

Pl. x, Fig. 2.

would not expect to find that absorption of dissepiments had taken place. If the theory of persistent germinal rudiments is to be accepted, we must suppose that some of these nuclea bodies which are so numerous in the foetal kidney near the periphery (*), have had their development arrested, that while the adjacent cellular structures have gone on to develop renal tubes, these have fallen out of line in different parts of the organ, that they have ceased to grow, and then at some future period they begin to multiply, to perform after a fashion the function for which they were originally adapted. It would also be natural to expect that if these cysts originated in this way we should have somewhere evidence of the commencement of the process in the form of small accumulations of epithelial elements, massed together in an irregular fashion, before the process had so far advanced as to deserve the name of cyst, this I have never seen although I have examined hundreds of sections.

*Sections 22+24

Those who support the theory that the cysts are new formations, whether from persistent germinal rudiments or otherwise, draw support to their views from the facts that there is great resemblance between liver and kidney cysts, and that as those which occur in the former organ contain only a clear fluid like glycerine, and not one having the appearance of bile, therefore they cannot be derived from bile ducts. Are they

correct in concluding that because the fluid is colorless it is not therefore altered bile? Virchow states that when bile is enclosed in this way three stages of change may be observed.

1) Accumulation of the bile in an obstructed duct.

2) Resorption of the elements of the bile, then secretion of mucus.

3) Serous transformation of the contents. Ritter and Prus hold that the colorless fluid found in the cysts is simply bile deprived of its coloring matter. That this is probably true is supported by a recent case recorded by Terrier(88), A woman, age 55, had for years suffered from symptoms of gall stone. On opening the abdomen a voluminous gall bladder was found, from which there was drawn off half a litre of clear, viscid, almost colorless fluid.

The second theory advanced is that the disease is an essential part of Cirrhosis, or that it is more or less intimately related to it.

Now, cirrhosis of the kidney is a common affection,whereas the large polycystic kidney is rare. In cirrhosis the cysts are mainly scattered on the surface, arising in the cortex, whereas in the large polycystic kidney, they are as frequent in the medulla as in the cortex. Colloid and other casts in the tubuli are common in cirrhosis without the development of cysts.

Laveran and others believe it to be independent of Interstitial Nephritis, because in affected kidneys there are considerable tracts which shew no increase of connective tissue, and others in which there is no heart enlargement. There is evidently an irritative lesion and the appearances of this are not confined to the areas in which the cysts abound, although they are usually most abundant there.

Simon states that there is a rupture of the limiting membrane of the tubes, an extravasation of the epithelial elements and their parenchytic development.

There is no evidence of the rupture of the membrana propria, and cysts arising from transplanted epithelial cells are usually solid with laminated contents consisting of piled up epithelium. The chief objection to this theory is that cysts can be seen in all stages of formation with a complete wall.

There is nothing to support Bouilland's theory that there is obstruction to the outflow, and a filtering of the retained fluid into the surrounding tissue, because the fluid is found in definite spaces with epithelial investment.

I have not found the appearances described by Dickinson, Cornil and Brault, also Kiener and Kelsch such as to justify the description of the process as 'a local destruction of tissue' or 'centres of degeneration', the cysts have in

general a well defined wall, but an examination of specimens Nos. (7297-73) will shew how such a mistake might arise. In some of the cysts in these sections there may be seen portions of tubules and also Malpighian bodies within the cyst, but a careful examination will shew that there is a very thin cyst wall, with little connective tissue, that the cyst wall has been complete, but has been ruptured in the process of cutting and mounting the section, and that, in consequence of the rupture, adjacent portions of kidney substance have been displaced.

I agree with those who consider the epithelial changes to be of primary importance, and accept in a measure the view of some.

The first question which naturally arises is as to whether the cyst formation is a transformation or a new growth. The presence of cysts does not necessarily involve the adoption of the letter view. The majority of observers who have expressed an opinion as to the cause of the cysts in Interstitial Nephritis, refer it to the cutting off or the obstruction of the orifices, of the Malpighian capsules, and the moniliform arrangement of the sacculations of the tubules they refer to the same cause. When discussing the views of those who believe that these cysts arise by a late development of persistent embryonic rudiments, it was pointed out that, if they arise in this way, we would expect to find somewhere evidences of the commencement

That is to say, these cysts are not supposed to be new growths.

of the process in the form of small accumulations of epithelial elements massed together, this I have never seen; secondly, that the pressure of diverticula - broad bands, or finger like projections into the cysts - point to the probability of the cysts arising in connection with urinary tubules. At the same time, having evidences of a budding process taking place, in at least some of the cases, at an early stage of the cyst formation I do not attach so much importance to this objection. It is evident however, that in cystic kidney these projections do not arise in consequence of the ingrowth of connective tissue, because they contain renal elements - tubes and bloodvessels. It has been already pointed out that many of these projections are not the remains of dissepiments which have been broken across in mounting, or which have been absorbed by pressure, seeing that, they have a complete epithelial investment, they occur in cysts of small size, and also that there is no counterfoil on the opposite side of the cyst. The other evidence is of a more positive character viz, that there are observed all stages of dilatation of tubules and of Malpighian bodies, and that in the latter there may occasionally be found remains of a glomerulus, further, that in many cases the position of a small cyst clearly points to its being the second of a pair of Malpighian bodies. I had expected, at first, more frequently to find the remains of a glomerulus if it were common for cysts

to arise by the cutting off and dilation of these corpuscles, but the examination of many specimens of cirrhosis (*) and of those of the advanced case of Hydronephrosis already described (**), has indicated the cause of the absence of the glomerulus. In these the glomerulus is seen in all stages of colloid transformation. In some also of the cystic kidneys the same change is seen.

*Sections 34 to 36

**Sections 3.4.5

Pl.II, Fig.1.

But is it possible that some of the cysts are a new growth, and that others, perhaps those with diverticula in them, are the result of obstruction, in consequence of pressure of the newly formed cysts on existing structures. Although it takes no great amount of lateral pressure to obstruct the flow of fluid through a tube, nevertheless we find evidence, both in connection with intra cranial tumors, and in cystic kidney, that the pressure of slowly growing bodies does not very seriously affect adjacent structures as regards their functions. In cystic kidney we find a large number of cysts, little apparent secreting substance, and that which remains subjected to a degree of pressure which, in some cases has completely distorted and displaced it, nevertheless almost up to the time of death the amount of fluid discharged has been above the average. In Hydronephrosis although there has been much pressure upon the secreting structure the dilatation is not in the capsules of Bowmann, nor in the tubules, but in the

pelvis and in the calyces. In the early stages, when the obstruction to the outflow of urine was not complete, the urine would first accumulate in the renal pelvis, but as pressure within a fluid is equal in all directions, the pressure within the tubules also must have been increased, the pressure would be exerted equally on the blood supply, and this is evidenced by the dilatation of veins and capillaries which is seen (* Sections 1 to 5), the secreting power would be lowered, and later the pressure would lead to the transformation of the elements of the kidney. What I wish to note is mainly, that the dilatation did not take place in the tubules and capsules, but in the pelvis and calyces. Further, the specimens of Hydronephrosis from a lamb and from a pig # shew how tolerant the renal structures are of pressure. # Sections 8 to 11 From these considerations it seems to me to be unlikely that cysts should arise in the kidney in consequence of pressure on the tubules by a new growth, leading to accumulation behind the obstruction.

Some of those who believe that cystic kidney of the adult arises in consequence of the retained embryonic rudiments, hold that the process is similar to that which is found in congenital cystic kidney. The probable causes of that affection are discussed at page 13 , the probability is that it arises in consequence of maldevelopment of the organs, and a failure of communication between the collecting tubules and the pelvis of

the kidney. For this reason as well as those formerly stated I do not believe that Cystic Kidney of the adult is the persistence of a congenital condition, nor that the cysts are produced in consequence of new growth apart from exicting secreting structures.

It has already been pointed out that, although cystic kidney presents many features in common with interstitial nephritis, there must be some difference seeing that cirrhosis is a condition frequently met with, whereas large polycystic kidney of the adult is rare; seeing that in contracting kidney the secreting tubules are much more atrophied and altered than those in cystic kidney which are found at a distance from cysts; seeing that although cysts are frequently found in Interstitial Nephritis they are usually small and not very abundant and the organs are consequently small, whereas the polycystic kidney is large with many large cysts. Although in the large cystic kidney there is in some regions much connective tissue,(a much greater development of it than is ever seen in contracting kidney) although it occurs in areas at a distance from cysts, leading to obliteration of tubes, nevertheless in the earlier stages there is not in it so diffuse a development.
In cystic kidney there is a bilateral tendency to cellular tissue and epithelial proliferation of a kind which does not occur in cirrhosis.

We cannot but be impressed by the fact, that in diseases producing sclerosis there is underlying them a constitutional tendency, that the same amount and kind of irritation will not in all constitutions produce interstitial new growth, and that where such changes do occur they are usually bilateral, they frequently affect many organs, and they exhibit paroxysmal exacerbations. The case of Ureteritis descendens, leading to Hydronephrosis, is a good example of some of these points. There occurred in my practise not long since a case which illustrates further the bilateral nature of affections of a similar nature accompanied by cellular proliferation leading to the stenosis of comparatively large canals. A lady age 45 consulted me in consequence of deafness in one ear, Dr. McBride found that the meatus had become completely occluded by a gradual stenosis of the meatus at some distance external to the membrana tympani, and on incising this new growth - which was of considerable thickness - he found a cavity separating it from the membrana tympani. A year later the other meatus was gradually narrowed, and eventually occluded in like manner, and that without the occurrence of any granulations, or discharge of any kind. Politzer has observed a few cases of a like kind. In this case there occurred not only an increase of interstitial tissue, but also epithelial proliferation.

Physiologically such increase of epithelium is not un-

common in certain glands, such as the mamma, in response to stimuli communicated through the nervous system. Such increase also takes place pathologically under a variety of conditions. In the mamma it is prone to occur at a time when the nervous system is in a mobile condition, when organs functionally related to it are in a state of change, and there are known causes of reflex irritation. In my experience cystic disease of the mamma occurs more frequently in the unmarried than in the married women, and chiefly about or soon after the climateric period. In proof of this there are subjoined short notes of the cases which have occurred in my practise.

Miss H. age 50 at the time of operation, had 8 years previous to operation noticed a very small lump in her left breast, although at first painless, after a time, when it had attained considerable dimensions, it caused much suffering. After removal it was found to consist of large and small cysts filled with Cholesterine and granular matter. After an interval of many years the other breast continued healthy.

Miss K. at age 48 had one mamma removed for cystic disease, it was of slow growth, and was troublesome in consequence of its size. Five years later the other breast was removed because of the same disease, it was also of slow growth.

Miss B. at age 41 had the right breast removed in consequence of a slow growth, which exhibited suspicious features.

There was no large tumor, but the whole organ was filled with a multitude of small cysts. Three years later the disease began to be well marked in the other breast, and it was ultimately removed.

The only two cases of Fibro-cystic tumor i.e. eucapsulated tumors, mainly fibrous with cysts, but not general cystic disease, occurred the one in a married lady age 39, the other in an unmarried lady age 29, and both in the left breast.

Stiles (90),who has devoted much attention to the development of cystic disease of the mamma,finds that the earliest part of the process is an increased development of the epithelium, a proliferation of it and a subsequent transformation.

Cystic disease of the mamma, and cystic kidney have these features in common, the time of life at which they most frequently occur, the bilateralism, proliferative changes in the epithelium and in the interstitial tissue.

It ought to be noted that subjects of cystic kidney have occasionally a similar disease in other organs, the liver, the ovary, the thyroid etc.

In sarcomata and in carcinomata there are degrees of rapidity of growth, and of those conditions which we call malignity so also in the adenomata we must recognise degrees of activity. Polymorphism of epithelium is characteristic of adenomata and the more columnar the cell the more active the proliferation.

So we must recognise in cystic kidney varieties of activity.

Can we offer any explanation of the differences in character of epithelium lining the cysts? Are we to explain this purely by the physical conditions within the cyst - mainly differences of pressure to which the epithelium is subjected -, or is there some other cause relating to cell activity and previous function? It is not to be denied that pressure does effect considerable changes, but that it is the sole or main cause is doubtful. On examining the cysts we find that the contents of those with squamous investment are mainly granular, and that those with columnar epithelium contain much colloid matter, and many cells which stain slightly, also nuclei which stain deeply. Those who hold the first theory will say that the columnar is still active undergoing proliferation, but that the squamous is subjected to greater pressure, that it is obsolete. We must remember that in the normal urinary apparatus although there can be no great difference of pressure, if any, in different parts of the same tube the epithelium varies considerably, being squamous in the capsule and varieties of cubical and columnar in the tubule. The difference between that of the capsule and tubule is seen even at a very early period of development in the foetal kidney (*). Epithelium may be squamous without being obsolete, but where great activity of function is required the epithelium is usually cubical or colum-

* Sections 17 to 21

nar. But the strongest proof that the question does not depend upon pressure is afforded by the fact that in the same cyst there may be seen different varieties, cubical and columnar, or squamous and cubical. The difference depends rather upon function and activity and I believe upon irritation direct or reflex; than upon pressure. In the urinary apparatus the watery part of the secretion is removed by the glomerulus, the solid constituents mainly by the epithelium of the tubule, and as the Malpighian body is the portion in which the watery elements are removed probably its epithelium has little functional activity.

*Pl. XI, Fig 2

Etiology in relation to Clinical History.

Although in a few individuals the disease has occurred at an earlier age - the earliest case being at the age of Five years - it is clear that it is in general an affection of middle or advanced life. The average age at which it occurs is about 45, rather earlier than the time at which cirrhosis is most frequent, but about the period at which cystic diseases of other organs is common. The clinical history much resembles that of cirrhosis, but with this difference, that there have been noted in cystic kidney marked paroxysmal attacks. These are common in cirrhotic maladies, as has been emphasized by Jaccoud both in relation to the liver, and more particularly

to the nervous system. In diseases of the cord, in consequence of the sensitiveness of the structures involved, the symptoms during the exacerbations are more evident than those of the congestive attacks affecting other organs. These congestive attacks in Cystic Kidney are characterized clinically by pains in the loins shooting up the back, and occasionally by haematuria. Cirrhosis is essentially an irritative lesion, the disease of the kidney is most frequent in those kinds of constitution in which the urine is loaded with morbid products. In Interstitial Nephritis as in cystic kidney the microscopic appearances give evidence of an irritative lesion, but in cystic kidney the clinical history shews more acute exacerbations - possibly local irritation acting through the nervous system causing congestive paroxysms. There is also evidence of more active proliferative changes in the epithelial elements than in Interstitial Nephritis, although in that affection they are not entirely absent.

The characters of the contents of cysts shew not only the activity of proliferation but that there has been extravasation into them,as well as haemorrhage externally. In the specimens shewn from advanced cases, the large glomeruli are of great interest in relation to the copious flow of urine.

Conclusions.

That in cystic kidney there is an irritative lesion leading to proliferation of epithelium and also to connective tissue changes.

That proliferation occurs in varying degree under different circumstances. There is evidence of it in simple cysts, in those which occur in contracting kidney, but to a much greater degree in the Large Polycystic Kidney. Possibly in the first two as a secondary effect, in the last primarily due to nerve influence.

That the cysts are formed from the pre-existing tubules and Malpighian bodies of the kidney, and that they are not new formations arising out of persistent embryonic rudiments.

That this cystic disease is related to and should be studied along with the Adenomata.

That cystic kidney is similar in character and in origin to cystic liver.

Diagnosis.

In early stages, in stout people, and with certain complications such as ascites and perinephritis, may be difficult or impossible. It is to be remembered that some people the subjects of this disease have made no complaint, and have not sought advice, and although they may have had symptoms, the first manifest indication has been the uraemic seizure

which has eventuated in death. Although there may have been no complaint, it is probable that there were symptoms, but not of such prominence as to cause the subject to seek advice. A similar experience is occasionally found in Interstitial Nephritis.

A history of paroxysmal attacks of pain in the lumbar region, shooting up the back, with or without haematuria, should lead to a careful examination of the renal regions. The presence of a lobulated tumor in each flank, a tumor which on palpation is found to be renal would be strong presumptive evidence of cystic disease of the kidney, all the more so if there were also present a copious flow of urine, the ordinary symptoms of Cirrhotic Bright's desease,urine copious, pale, of low sp. gr. and with a trace of albumen. Even with these symptoms the diagnosis would not be certain. At the commencement of this paper there is recorded the history of a case of Hydronephrosis due to Ureteritis descendens, which was diagnosed as cystic kidney, because Hydronephrosis is usually unilateral, unless in kidneys with calculi, or when there is present one of the ordinary causes of Hydronephrosis, such as bladder or uterine mischief or pelvic tumor. If there are calculi the pain is more severe than in cystic kidney, and it often shoots downwards in the line of the ureter.

Cancer and sarcoma usually affect only one side. There is

more frequent haematuria, but there are not the symptoms of cirrhosis, and the tumor is rounder - not so nodulated.as in cystic kidney, and the quantity of urine is not increased.

In all cases the bimanual method of palpation is indispensible, without this it is impossible to differentiate hepatic or splenic tumors from above, and from below ovarian and uterine, as well as other abdominal growths.

From Ovarian tumors the diagnosis has been fully given by Spencer Wells (30) of this I shall give an epitome.

1) Apart from rare exceptions ovarian tumors push the intestine behind, the kidneys have it in front.

2) Large tumors of right kidney have the ascending colon on on their internal border,- of left kidney crossed from above downwards by the descending colon.

3) The existence of intestine in front of an abdominal tumor calls for examination of the urine. It is possible that the urine may be healthy even if the kidney is affected, but usually blood, pus, albumen, or renal epithelium will be present.

4) Percussion may not be sufficient to determine the presence of the intestine, but on palpation the intestine gives the idea of a thick pad under the fingers.

5) Both renal and ovarian cysts may be liable to great alterations in size. If fluid escapes by the bladder, rectum, or vagina, its characters should be tested.

6) If a correct history can be obtained the renal tumor begins near the false ribs, the ovarian in the inguinal and iliac regions extending from below upwards.

7) Pedunculated ovarian tumors might be mistaken for moveable kidney, but the form of tumor and the percussion note over the lumber region help.

8) Renal tumors are usually associated with renal symptoms, ovarian with disturbances of menstruation, pain at catamenial periods but there are cases of both in which no abnormality of function can be discovered.

Difficulty in diagnosis of cystic kidney is most likely to be experienced in the early stages, but a history of paroxysmal pain, haematuria, and the pressure of nodulated tumor should excite suspicion. In the later stages the characters of the urine, specially a copious discharge of it, the cachexia, and the presence of nodular tumors on both sides, which are renal, should render the diagnosis tolerably clear.

Prognosis.

Is unfavorable. Several cases which have been discovered in a comparatively early stage have been under observation for several years. The prognosis will be guided largely by the condition of nutrition, the character of the urinary secretion, and the rate at which the disease has been progressing, when

this is known.

Treatment.

Should be that adopted in cases of cirrhosis of kidney, careful dieting, warmth to the surface, and guarding against chills. Operative treatment has been adopted, and in the case by Thiriar, recovery after operation is recorded, but considering the bilateral nature of the disease operation is practically excluded.

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